EUROPEAN RESUSCITATION COUNCIL

Advanced Life Support

ERC GUIDELINES 2015 EDITION

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Advanced Life Support
Course Manual

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Glossary

ACEI	Angiotensin Converting Enzyme Inhibitors
ACS	Acute Coronary Syndrome
AED	Automated External Defibrillator
AF	Atrial Fibrillation
ALS	Advanced Life Support
AMI	Acute Myocardial Infarction
A-P	anterior-posterior
ARB	Angiotensin II Receptor Blockers
ARDS	Acute Respiratory Distress Syndrome
ASA	Acetylsalicylic acid
AV	Atrioventricular
AVNRT	AV Nodal Re-entry Tachycardia
AVRT	AV Re-entry Tachycardia
BE	Base Excess
BLS	Basic Life Support
BMV	Bag-Mask Ventilation
BP	Blood Pressure
CA	Cardiac Arrest
CABG	Coronary Artery Bypass Grafts
CAS	Cardiac Arrest Simulation
CHB	Complete Heart Block
CK	Creatin kinase
CO$_2$	Carbon Dioxide
COPD	Chronic Obstructive Pulmonary Disease
CPP	Cerebral Perfusion Pressure
CPR	Cardiopulmonary Resuscitation

CRT	Capillary refill time
CV ratio	Compression Ventilation Ratio
DNAR	Do Not Attempt Resuscitation
ECG	Electrocardiogram
ECLS	Extracorporeal Life Support
ED	Emergency Department
EEG	Electroencephalogram
EMS	Emergency Medical Services
ETCO$_2$	End-Tidal Carbon Dioxide
EWS	Early warning score
FBAO	Foreign Body Airway Obstruction
F$_I$O$_2$	Inspired Oxygen Fraction
GCS	Glasgow Coma Scale
GRACE	Global Registry of Acute Coronary Events
HDU	High Dependency Unit
HR	Heart Rate
IABP	Intra Aortic Balloon Pump
ICD	Implantable Cardioverter-defibrillator
ICP	Intracranial Pressure
ICU	Intensive Care Unit
ID	Internal Diameter
IN	intranasal
IO	intraosseous
IV	intravenous
J	Joule
J/Kg	Joules Per Kilogram

JVP	Jugular Venous Pressure	**PEA**	Pulsless Electrical Activity
L/Min	Liters Per Minute	**PEEP**	Positive End Exspiratory Pressure
LBBB	Left Bundle Branch Block	**PEF**	Peak Expiratory Flow
LT	Laryngeal tube	**PRC**	Packed Red Cells
LV	Left Ventricle	**pVT**	Pulsless ventricular Tachycardia
MAP	Mean Arterial Pressure	**ROSC**	Return of Spontaneous Circulation
Mcg/Kg	Microgram Per Kilogram		
MET	Medical Emergency Team	**RR**	Respiratory Rate
MI	Myocardial Infarction	**RSI**	Rapid Sequence Intubation
Ml/H	Milliliters Per Hour	**RSVP**	Reason Story Vital Signs Plan
Ml/Kg	Milliliters Per Kilogram	**RV**	Right Ventricle
MRI	Magnetic resonance imaging	**SBAR**	Situation Background Assessment Recomendation
NHS	National Health Service		
NIRS	Near infra-red spectroscopy	**SC**	subcutaneous
NPSA	National Patient Safety Agency	**SCA**	Sudden Cardiac Arrest
NSAIDs	Non-steroidal Anti-inflammatory Drugs	**SGA**	Supraglottic airway
		SPECT	Single photon emission computed tomography
NSTEMI	Non-ST-segment-elevation myocardial infarction		
		SpO_2	Peripheral Oxygen Saturation
NTS	Non Technical Skills	**SVT**	Supraventricular Tachycardia
O_2	Oxygen	**TCA**	Traumatic Cardiac Arrest
OHCA	Out-Of-Hospital Cardiac Arrest	**TTM**	Targeted temperature management
$PaCO_2$	Partial Arterial Carbon Dioxide Pressure		
		TXA	Tranexamic Acid
PAD	Public Access Defibrillation	**VF**	Ventricular Fibrillation
PaO_2	Partial Arterial Oxygen Pressure		
PCI	Percutaneous Coronary Intervention		

Chapter 1.

Advanced Life Support in perspective

1.   The problem

Ischaemic heart disease is the leading cause of death in the world. In Europe sudden cardiac arrest (SCA) is one of the leading causes of death. Depending how SCA is defined, about 55-113 per 100,000 inhabitants a year or 350,000-700,000 individuals a year are affected in Europe. On initial heart-rhythm analysis, about 25-50 % of SCA victims have ventricular fibrillation (VF), a percentage that has declined over the last 20 years. It is likely that many more victims have VF or rapid ventricular tachycardia (VT) at the time of collapse, but by the time the first electrocardiogram (ECG) is recorded by emergency medical service personnel their rhythm has deteriorated to asystole. When the rhythm is recorded soon after collapse, in particular by an on-site AED, the proportion of victims in VF can be as high as 76 %. More victims of SCA survive if bystanders act immediately while VF is still present. Successful resuscitation is less likely once the rhythm has deteriorated to asystole. The incidence of treated OHCAs was higher in North America (54.6) than in Europe (35.0), Asia (28.3) and Australia (44.0) (p< 0.001). In Asia, the percentage of VF and survival to discharge rates were lower (11 % and 2 %, respectively) than those in Europe (35 % and 9 %, respectively), North America (28 % and 6 %, respectively), or Australia (40 % and 11 %, respectively).

One third of all people developing a myocardial infarction die before reaching hospital; most of them die within an hour of the onset of acute symptoms. In most of these deaths the presenting rhythm is VF or pulseless ventricular tachycardia (VF/pVT). The only effective treatment for these arrhythmias is attempted defibrillation and, in the absence of bystander CPR, with each minute's delay the chances of a successful outcome decrease by about 10-12 %. Once the patient is admitted to hospital the incidence of VF after myocardial infarction is approximately 5 %.

The incidence of in-hospital cardiac arrest is difficult to assess because it is influenced heavily by factors such as the criteria for hospital admission and implementation of a do-not-attempt-resuscitation (DNAR) policy. The reported incidence of in-hospital cardiac arrest is in the range of 1-5 per 1000 admissions. Data from the UK National Cardiac Arrest Audit (NCAA) indicate that survival to hospital discharge after in-hospital cardiac arrest is 13.5 % (all rhythms). The initial rhythm is VF or pulseless VT in 18 % of cases and, of these, 44 % survive to leave hospital; after PEA or asystole, 7 % survive to hospital discharge. These preliminary NCAA data are based on 3,184 adults (aged ≥ 16 y) in 61 hospitals participating in NCAA (increasing numbers of hospitals during Oct 2009 to Oct 2010) with known presenting/first documented rhythm and complete data for return of spontaneous circulation (ROSC)

and survival to hospital discharge. All these individuals received chest compressions and/or defibrillation from the resuscitation team in response to a 2222 call. Many in-hospital cardiac arrests did not fulfil these criteria and were not included. Many patients sustaining an in-hospital cardiac arrest have significant comorbidity, which influences the initial rhythm and, in these cases, strategies to prevent cardiac arrest are particularly important.

1.2. The Chain of Survival

The interventions that contribute to a successful outcome after a cardiac arrest can be conceptualised as a chain – the Chain of Survival *(figure 1.1)*. The chain is only as strong as its weakest link; all four links of the Chain of Survival must be strong. They are:

- early recognition and call for help
- early cardiopulmonary resuscitation (CPR)
- early defibrillation
- post-resuscitation care

Figure 1.1
Chain of survival
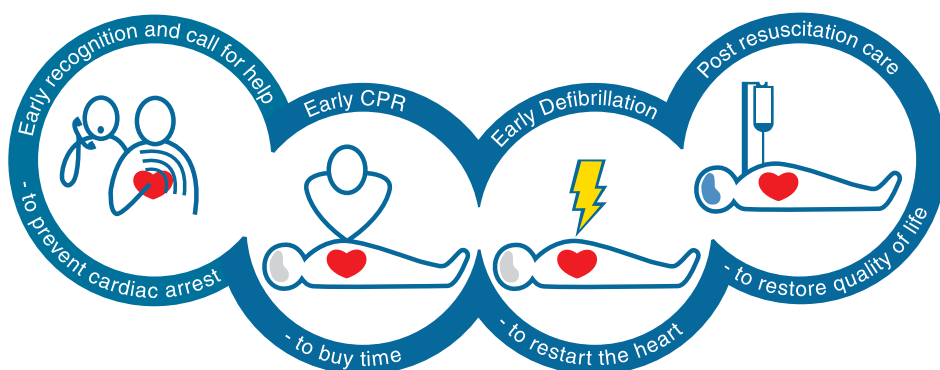
1.2.1. Early recognition and call for help

Out of hospital, early recognition of the importance of chest pain will enable the victim or a bystander to call the EMS so that the victim can receive treatment that may prevent cardiac arrest. After out-of-hospital cardiac arrest, immediate access to the EMS is vital. In most countries access to the EMS is achieved by means of a single telephone number (e.g. 112, 999).

In-hospital, early recognition of the critically ill patient who is at risk of cardiac arrest and a call for the resuscitation team or medical emergency team (MET) will enable treatment to prevent cardiac arrest *(chapter 3)*. A universal number for calling the resuscitation team or MET should be adopted in all hospitals. If cardiac arrest occurs, do not delay defibrillation until arrival of the resuscitation team. The clinical staff should be trained to use a defibrillator.

1.2.2. Early CPR

Chest compressions and ventilation of the victim's lungs will slow down the rate of deterioration of the brain and heart. After out-of-hospital cardiac arrest, bystander CPR extends the period for successful resuscitation and at least doubles the chance of survival after VF cardiac arrest. Performing chest-compression-only CPR is better than giving no CPR at all. Despite the well-accepted importance of CPR, in most European countries bystander CPR is carried out in only a minority of cases (approximately 30 %). After in-hospital cardiac arrest, chest compressions and ventilation must be undertaken immediately, but should not delay attempts to defibrillate those patients in VF/pVT. Interruptions to chest compressions must be minimised and should occur only very briefly during defibrillation attempts and rhythm checks.

1.2.3. Early defibrillation

After out-of-hospital cardiac arrest, the goal is to deliver a shock (if indicated) within 5 min of the EMS receiving the call. In many areas, achievement of this goal will require the introduction of Public Access Defibrillation (PAD) programs using automated external defibrillators (AEDs). In hospitals, sufficient healthcare personnel should be trained and authorised to use a defibrillator to enable the first responder to a cardiac arrest to attempt defibrillation when indicated, without delay, in virtually every case.

1.2.4. Post-resuscitation care

Return of a spontaneous circulation (ROSC) is an important phase in the continuum of resuscitation; however, the ultimate goal is to return the patient to a state of normal cerebral function, a stable cardiac rhythm, and normal haemodynamic function, so that they can leave hospital in reasonable health at minimum risk of a further cardiac arrest. The quality of treatment in the post-resuscitation period influences the patient's ultimate outcome. The post-resuscitation phase starts at the location where ROSC is achieved. The ALS provider must be capable of providing high-quality post-resuscitation care until the patient is transferred to an appropriate high-care area.

1.3.　Science and guidelines

The 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations was the culmination of a prolonged period of collaboration between resuscitation experts from around the world. It followed a similar format to the 2010 International Consensus on CPR Science. The European Resuscitation Council (ERC) Guidelines for Resuscitation 2015 are derived from the 2015 consensus document and the contents of this ALS provider manual are consistent with these guidelines. Most resuscitation organisations in Europe have ratified and adopted the ERC guidelines.

1.4.   ALS algorithm

The ALS algorithm *(figure 1.2)* is the centre point of the ALS course and is applicable to most cardiopulmonary resuscitation situations. Some modifications may be required when managing cardiac arrest in special circumstances *(chapter 12)*.

1.5.   The ALS course

The ALS course provides a standardised approach to cardiopulmonary resuscitation in adults. The course is targeted at doctors, nurses, and other healthcare professionals who are expected to provide ALS in and out of hospital. The multidisciplinary nature of the course encourages efficient teamwork. By training together, all ALS providers are given the opportunity to gain experience as both resuscitation team members and team leaders.

The course comprises workshops, skill stations, cardiac arrest simulation (CAS) training, and lectures. Candidates' knowledge is assessed by means of a multiple choice questions. Practical skills in airway management and the initial approach to a collapsed patient (including basic life support and defibrillation where appropriate) are assessed continuously.

There is also assessment of a simulated cardiac arrest (CASTest). Candidates reaching the required standard receive an ALS provider certificate. Resuscitation knowledge and skills deteriorate with time and therefore recertification is required. Recertification provides the opportunity to refresh resuscitation skills and to be updated on resuscitation guidelines, and can be undertaken by attending a provider course or an accredited recertification course. All ALS providers have a responsibility to maintain their skills in resuscitation and to keep up to date with changes in guidelines and practice, and the requirement for recertification should be seen as an absolute minimum frequency of refreshing skills and knowledge.

Figure 1.2.
Adult Advanced Life Support

**Unresponsive and
not breathing normally?**

Call Resuscitation Team

CPR 30:2
Attach defibrillator/monitor
Minimise interruptions

Assess rhythm

**Shockable
(VF/Pulseless VT)**

**Non-shockable
(PEA/Asystole)**

1 Shock
Minimise
interruptions

**Return of
spontaneous
circulation**

Immediately resume
CPR for 2 min
Minimise interruptions

**IMMEDIATE POST
CARDIAC ARREST
TREATMENT**
- Use ABCDE approach
- Aim for SpO_2 of 94-98 %
- Aim for normal $PaCO_2$
- 12 Lead ECG
- Treat precipitating cause
- Targeted temperature
  management

Immediately resume
CPR for 2 min
Minimise interruptions

DURING CPR
- Ensure high-quality chest compressions
- Minimise interruptions to compressions
- Give oxygen
- Use waveform capnography
- Continuous compressions when advanced
  airway in place
- Vascular access
  (intravenous or intraosseous)
- Give adrenaline every 3-5 min
- Give amiodarone after 3 shocks

TREAT REVERSIBLE CAUSES

Hypoxia	Thrombosis – coronary or pulmonary
Hypovolaemia	Tension pneumothorax
Hypo-/hyperkalaemia/metabolic	Tamponade – cardiac
Hypothermia/hyperthermia	Toxins

CONSIDER
- Ultrasound imaging
- Mechanical chest compressions to facilitate transfer/treatment
- Coronary angiography and percutaneous coronary intervention
- Extracorporeal CPR

FURTHER READING

- Perkins G et al. European Resuscitation Council Guidelines for Resuscitation 2015
- Section 2: Adult basic life support and automated external defibrillation 10.1016/j.resuscitation.2015.07.015; p81 - p98
- Soar J et al. European Resuscitation Council Guidelines for Resuscitation 2015
- Section 3. Adult advanced life support 10.1016/j.resuscitation.2015.07.016; p99 - p146
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Chapter 2.

Non-technical skills and quality in resuscitation

> **LEARNING OUTCOMES**
>
> **To understand:**
> - the role of human factors in resuscitation
> - how to use structured communication tools such as SBAR and RSVP
> - the role of safety incident reporting and audit to improve patient care

1.   Introduction

Skills such as defibrillation, effective chest compressions, ability to ventilate, recognition of the underlying cardiac arrest rhythm, which are all important components of successful resuscitation, are usually termed technical skills. These skills are acquired from many different sources, including courses. Despite the fact that there is little disagreement that these skills are necessary for human resuscitation, recently another category of skills and factors emerged. The terms human factors and non-technical skills have been used interchangeably, yet each has a specific definition. Non-technical skills are the cognitive and interpersonal skills that underpin effective team work and it is estimated that between 70-80 % of healthcare error(s) can be attributed to a breakdown in these skills. Non-technical skills include the interpersonal skills of communication, leadership and followership (being a team member) and the cognitive skills of decision making, situation awareness and task management. Non-technical skills are part of the human factors agenda. Human factors is an umbrella term which analyses how healthcare professionals interact with everything in their working environment, such as clinical guidelines, policies and procedures, equipment and stress management. It also encompasses the improvement of day-to-day clinical operations, through an appreciation of the effects of teamwork on human behaviour and the application in a clinical setting. Non-technical skills specifically examine the interaction of team members, leaving all other elements of the human factors aside. Both non-technical skills and human factors are starting to be recognized as equally important in resuscitation medicine, but are often poorly articulated in formal courses, during hospital training and during any assessment.

Introduction and formal training in human factors and non-technical skills has led to a significant reduction of accidents of aviation and it was only recently that medicine acknowledged the importance of these skills. There is little doubt that the pioneers in this

were the anaesthesiologists with the development of formal courses but surgeons and other specialties started to take interest in these skills. Historically, however, it was Leape that pointed that errors in medicine do happen because of poor communication, and one cross-sectional survey associated error, stress and teamwork in medicine and aviation. There is little doubt that both aviation and resuscitation medicine are professions where errors can have detrimental consequences. Despite the advances in aviation safety, a very modern aircraft crashed in 2009, killing more than 200 passengers. The analysis of the errors during this flight has revealed that the pilots in this flight operated under severe stress and it comes as no surprise that all cardiac arrest teams often operate under a lot of stress. Interestingly, the similarities and the lessons drawn from the fatal flight 447 for medicine were done by surgeons.

There are several systems that have been developed to ensure acceptable use of non-technical skills, such as the team dimension rating form, the Oxford Non-Technical Skills Measure, just to name a few. The principles used to promote good non-technical skills in the ALS course are based on Team Emergency Assessment Measure. The proposed Taxonomy in NTS, adopted by the ERC is illustrated in table 2.1.

2. Leadership

In reality leadership is an attribute that is extremely difficult to define. Various scholars have defined leadership in various ways. The definitions agree that an effective leader is the person with a global perspective of the situation s/he is facing and as a result s/he allocates roles to various team members in order to accomplish the global perspective of the leader. Medical literature agrees that leadership is not a trait, but it can be accomplished with continuous training. In resuscitation teams, the team leader needs to:

1. Let the team know exactly what is expected from them. This entails a high level of situational awareness, an ability to allocate tasks according to the team-members' experience, establishes his/her decision making process using evidence based medicine and clearly verbalising his/her decisions. A good team leader always knows and addresses his/her team by name and can act as a role model for the team to evolve.

2. Maintain a high level of global perspective. In reality this means putting a plan that the leader has into action. As team-members perform their tasks, the team leader carefully monitors whether these are being performed. In the setting of cardiopulmonary resuscitation, the team leader should always be able to hear what information the team-members are relaying to him/her. Consecutively, the leader should be able not just to monitor the clinical procedures as they are performed, but also to be able to provide guidance as the procedures occur, remaining "hands free". Safe practice of all procedures is the responsibility of the leader, not just for the patient, but for the team of healthcare professionals working together. The team leader should also be empathic to other healthcare professionals and should possess inter-professional communication skills.

3. Successful planning. During CPR, the team leader should be able to plan the next actions either by filtering the available data or by anticipating the most possible scenarios. Team readiness and rapid execution of actions ordained by the leader are essential elements for ensuring high-quality CPR.

Table 2.1.

Taxonomy of Non-Technical Skills adapted and modified from Cooper et al (2010) to be used in ALS training courses. See *http://medicalemergencyteam.com/* for full details

2

LEADERSHIP	Not seen (√)	Observed (√)
The team leader let the team know what was expected of them through direction and command. **Examples:** uses members names, allocates tasks, makes clear decisions		
The team leader maintained a global perspective. **Examples:** monitors clinical procedures, checks safety, plans ahead, remains 'hands off'		
TEAMWORK		
The team communicated effectively, using both verbal and non-verbal communication. **Examples:** relay findings, raise concerns, use names, appropriate body language		
The team worked together to complete tasks in a timely manner. **Examples:** coordination of defibrillation, maintain chest compressions, assist each other		
The team acted with composure and control. **Examples:** performed allocated roles, accept criticism		
The team adapted to changing situations. **Examples:** adapt to rhythm changes, patient deterioration, change of roles		
The team monitored and reassessed the situation **Examples:** rhythm changes, ROSC, when to terminate resuscitation		
The team anticipated potential actions. **Examples:** defibrillation, airway management, drug delivery		
TASK MANAGEMENT		
The team prioritised tasks. **Examples:** continuous chest compressions, defibrillation, airway management, drug delivery		
The team followed approved standards/guidelines.		
COMMENTS		
Examples: What area was good? What area needs improvement?		

3. Teamwork

Teamwork is one of the most important NTS that can contribute to the management of a cardiac arrest patient. Clinical competence and clinical experience are important for the outcome of resuscitation, but not a guarantee of success. As with leadership, teamwork has to be learnt and practiced in various settings to enhance resuscitation team performance. A team is a group of healthcare professionals with different skills and different backgrounds working together to achieve a common goal. The leader is an integral part of the team, but each team member is equally important in the team performance. The key elements for effective team performance are:

1. Effective verbal and non-verbal communication. The team needs to relay the findings as they occur and they should be able to understand what the leader's plan is, carrying out of the allocated tasks and always closing the communicational loop. The team members should be able to raise concerns, but they should always filter the information they provide. In cardiac arrest management, several things are happening simultaneously and effective communication needs practice within this setting. The ALS course is such a training opportunity for teams to practice effective communication.

2. Working together to complete tasks in a timely manner. Time is important in CPR and team co-ordination is extremely important for the safe delivery of defibrillation, as well as maintaining high-quality chest compressions throughout the resuscitation attempt. Cardiopulmonary resuscitation is a stressful situation during which suboptimal individual performance may affect team's performance, especially when the leader fails to fulfil his/her role. Each team member should, in a polite manner and in the context of a harmonious cooperation, draw attention either directly to the failing team member or, better, to inform the leader avoiding any potential infringement of other members.

3. Acting with composure and control. There are many internal and external factors affecting the team structure. Each team member following the allocated roles characterizes effective teamwork. Conflict management and an adequate criticism culture are important instruments enabling good team performance.

4. Adaptation to changing situations. Cardiac arrest management is a dynamic procedure. Cardiac arrest patients are by defition extremly unstable even when they achieve Return of Spontaneous Circulation (ROSC). During CPR the team should be comfortable to changing roles (eg alternation of the airway person with the compressor) and they should be able to adapt to rhythm changes, when these occur. In addition, adaptation to scene is crucial for optimizing high-quality care. Team members should be able to adapt to any cardiac arrest scene, including the ICU, ER, or in a non-spacious ward room.

5. Reassessment of the situation. In CPR this means not only continuous reassessment of the patient but also a consensus on when resuscitation attempts should be terminated. The ERC guidelines provide clear guidance on when resuscitation should be terminated.

6. Anticipation of potential actions. In CPR this entails preparation for airway management, preparation and delivery of drugs, preparation and energy selection for defibrillation.

4. Task management

During the resuscitation of any patient, either in the peri-arrest or full cardiac arrest situation, there are numerous tasks that should be carried out by the team. These include:

1. Prioritization of the tasks that should be performed either simultaneously or sequentially. This skill includes defining tasks and organizing them by priority and sequence. Knowledge and effective use of the resources available are additional important factors.

2. Adhering to current and approved guidelines and practices. This includes deviations where appropriate.

3. Ensuring high-quality post-resuscitation care and timely patient transport either to the catheterization laboratory or the intensive care unit. The team members must be skilled enough to continue post-resuscitation treatment in different settings, including the ICU, until they deliver the patient to specialized personnel.

Figure 2.1
Task management

5. Audit and registry reporting

Regarding CPR, hospital-level infrastructure may comprise a coordinating resuscitation committee and resuscitation teams. This enables periodic, multilevel, clinical cardiac arrest audit aimed at continuous improvement of the resuscitation service. The audit pertains to the availability and use of resuscitation/peri-arrest drugs and resuscitation equipment, the always prompt activation in case of in-hospital cardiopulmonary arrest, the documentation of management using the Utstein template and relevant audit forms, do-not-attempt

resuscitation decisions and policies, outcomes, critical incidents leading to or occurring during CPR, and various safety/logistical issues (e.g. decontamination/maintenance of training/resuscitation equipment).

Local CPR management can be improved through post-CPR debriefing aimed at determining CPR quality errors and mitigating their repetition during subsequent CPR attempts. Examples of such errors include low-rate and/or shallow chest compressions, prolonged interruptions of chest compressions, and excessive ventilation. Institutions should also be encouraged to submit CPR data in standardized format to national audits and/or international registries aimed at continuous quality improvement. Such practices have already led to the development of validated outcome-predictive models, which may facilitate advanced care planning. In addition, a prior registry data analysis quantified the frequency of resuscitation system errors and their impact on in-hospital mortality after shockable and non-shockable cardiac arrest. Registry results have shown significant improvements in cardiac arrest outcomes within 2000-2010.

Published evidence suggests that resuscitation team-based infrastructure, multilevel institutional audit, accurate reporting of resuscitation attempts at national audit level and/or multinational registry level, and subsequent data analysis and feedback from reported results may contribute to continuous improvement of in-hospital CPR quality and cardiac arrest outcomes.

6. The importance of communication when managing a sick patient

Communication includes seeking and reporting information. During CPR, communication between team members can be verbal and non-verbal, as well as informal and structured. Of note, cardiac arrest teams may face many kinds of communication challenges at the professional, organizational, team, personal and/or patient, levels which may affect the quality of CPR.

Effective teamwork and communication skills are critical success factors during CPR; poor communication will decrease team's effectiveness and survival rates. This usually happens due to inconsistency of team members from day to day which seriously affects communication skills. Consequently, optimization of team communication can be optimized through high-quality training, during which concepts and applications for effective team communication can be implemented, focusing on several approaches, team interaction, and relationship management.

Individual team members, regardless of the member's position, must learn to perceive orders and accept their roles as non-intimidating. Team orientation should be built by taking steps to increase trust and cohesion and increase satisfaction, commitment, and collective efficacy. Although increasing awareness of different communication styles and possibly incorporating these skills into medical training may help teams arrive more efficiently at jointly managed efforts during CPR, precise and accurate communication through a closed-loop communication protocol should be always encouraged. Use of SBAR

during written and verbal communication, active listening, body language, and tone of voice may also help team members to recognize and understand individual personal styles, preferences and temperament types. Appreciating others' differences will enhance the approach between team members and increase team efficiency.

Table 2.2.
SBAR and RSVP communication tools

SBAR	RSVP	Content	Example

Situation	**R**eason	• Introduce yourself and check you are speaking to the correct person.
		• Identify the patient you are calling about (who and where).
		• Say what you think, the current problem is, or appears to be.
		• State what you need advice about.
		• Useful phrases:
		  - The problem appears to be cardiac/respiratory/neurological/sepsis.
		  - I'm not sure what the problem is but the patient is deteriorating.
		  - The patient is unstable, getting worse and I need help.
			• Hello, I am Dr Smith the junior medical doctor.
			• I am calling about Mr Brown on acute medical admissions who I think has a severe pneumonia and is septic.
			• He has an oxygen saturation of 90 % despite high-flow oxygen and I am very worried about him

Background	**S**tory	• Background information about the patient • Reason for admission • Relevant past medical history	• He is 55 and previously fit and well. • He has had fever and a cough for 2 days. • He arrived 15 minutes ago by ambulance.

SBAR	RSVP	Content	Example
Assessment	**V**ital Signs	• Include specific observations and vital sign values based on ABCDE approach: • Airway • Breathing • Circulation • Disability • Exposure • The early warning score is…	• He looks very unwell and is becoming tired. • Airway - he can say a few words. • Breathing - his respiratory rate is 24, he has bronchial breathing on the left side. His oxygen saturation is 90 % on high-flow oxygen. I am getting a blood gas and chest X-ray. • Circulation - his pulse is 110, his blood pressure is 110/60. • Disability - he is drowsy but can talk a few words. • Exposure - he has no rashes.
Recommendation	**P**lan	• State explicitly what you want the person you are calling to do. • What by when? • Useful phrases: - I am going to start the following treatment; is there anything else you can suggest? - I am going to do the following investigations; is there anything else you can suggest? - If they do not improve; when would you like to be called? - I don't think I can do any more; I would like you to see the patient urgently	• I am getting antibiotics ready and he is on IV fluids. • I need help - please can you come and see him straight away.

7. High-quality care

Quality care can be described as safe, effective, patient-centred, timely, efficient and equitable. Hospitals, resuscitation teams and ALS providers should ensure they deliver these aspects of quality to improve the care of the deteriorating patient and patients in cardiac arrest. Two aspects of this are safety incident reporting (also called adverse or critical incident reporting) and collecting good quality data.

7.1. Safety incident reporting

There are a number of critical incident reporting systems throughout Europe. For example, in England and Wales hospitals can report patient safety incidents to the National Patient Safety Agency (NPSA) National Reporting and Learning System (NRLS) *(http://www.nrls. npsa.nhs.uk/report-a-patient-safetyincident/).*

A patient safety incident is defined as "any unintended or unexpected incident that could have harmed or did lead to harm for one or more patients being cared for by the National Health Service (NHS)". Previous reviews of this database have identified patient safety incidents associated with airway devices in critical care units and led to recommendations to improve safety. A review of NPSA safety incidents relating to cardiac arrest and patient deterioration by the Resuscitation Council (UK) shows that the commonest reported incidents are associated with equipment problems, communication, delays in the resuscitation team attending, and failure to escalate treatment.

8. Audit and outcome after cardiac arrest

Measurement of processes and outcomes provides information about whether interventions and changes made to resuscitation guidelines improve patient care.
Published survival rates from in-hospital cardiac arrest vary substantially and range from 13-59 % at 24 h and 3-27 % to discharge, with a median survival to discharge of about 15 %. There are probably two main reasons for such variation: firstly, there are many confounders that influence outcome following cardiac arrest. These include:

- differences in the type of EMS system (e.g. availability of defibrillators, differences in response intervals)

- differences in the incidence of bystander CPR

- different patient populations (e.g. a study may be confined to in-hospital cardiac arrests or may include pre-hospital arrests)

- the prevalence of co-morbid conditions

- the frequency of implementing do-not-attempt resuscitation (DNAR) policies

- the primary arrest rhythm

- the definition of cardiac arrest (e.g. inclusion of primary respiratory arrests)

- availability of cardiac arrest and medical emergency teams

Secondly, there is lack of uniformity in reporting both the process and results of resuscitation attempts; for example, the definition of survival is reported variously as return of spontaneous circulation, or survival at 5 min, 1 h, 24 h, or to discharge from hospital. The lack of uniformity in cardiac arrest reporting makes it difficult to evaluate the impact on survival of individual factors, such as new drugs or techniques.

New interventions that improve survival rate only slightly are important because of the many victims of cardiac arrest each year. Local hospitals or healthcare systems are unlikely to have sufficient patients to identify these effects or eliminate confounders. One way around this dilemma is by adopting uniform definitions and collecting standardised data on both the process and outcome of resuscitation on many patients in multiple centres.

Changes in the resuscitation process can then be introduced and evaluated using a reliable measure of outcome. This methodology enables drugs and techniques developed in experimental studies to be evaluated reliably in the clinical setting.

Most European countries have a national audit for in- and out-of-hospital cardiac arrests. These audits monitor and report on the incidence of, and outcome from, cardiac arrest in order to inform practice and policy. They aim to identify and foster improvements in the prevention, care delivery and outcomes from cardiac arrest.

Data are usually collected according to standardised definitions and entered onto secure web-based systems.
Once data are validated, participants are provided with activity reports and comparative reports, allowing a comparison to be made not only within, but also between, systems locally, nationally and internationally.
Furthermore it also enables the effects of introducing changes to guidelines, new drugs, new techniques etc to be monitored that would not be possible on a single participant basis.

> **KEY LEARNING POINTS**
>
> - Human factors are important during resuscitation.
> - Use SBAR or RSVP for effective communication.
> - Report safety incidents and collect cardiac arrest data to help improve patient care.

2

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Chapter 3.

Recognition of the deteriorating patient and prevention of cardiorespiratory arrest

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LEARNING OUTCOMES

- the importance of early recognition of the deteriorating patient
- the causes of cardiorespiratory arrest in adults
- how to identify and treat patients at risk of cardiorespiratory arrest using the Airway, Breathing, Circulation, Disability, Exposure (ABCDE) approach

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1.   Introduction

Early recognition of the deteriorating patient and prevention of cardiac arrest is the first link in the chain of survival. Once cardiac arrest occurs, fewer than 20 % of patients having an in-hospital cardiac arrest will survive to go home. Prevention of in-hospital cardiac arrest requires staff education, monitoring of patients, recognition of patient deterioration, a system to call for help, and an effective response.

Survivors from in-hospital cardiac arrest usually have a witnessed and monitored ventricular fibrillation (VF) arrest, primary myocardial ischaemia as the cause, and receive immediate and successful defibrillation.

Most cardiorespiratory arrests in hospital are not sudden or unpredictable events: in approximately 80 % of cases there is deterioration in clinical signs during the few hours before cardiac arrest. These patients often have slow and progressive physiological deterioration, particularly hypoxia and hypotension (i.e. Airway-Breathing-Circulation problems) that is unnoticed by staff, or is recognised but treated poorly. The cardiac arrest rhythm in this group is usually non-shockable (PEA or asystole) and the survival rate to hospital discharge is very low.

Early recognition and effective treatment of the deteriorating patient might prevent cardiac arrest, death or an unanticipated intensive care unit (ICU) admission. Closer attention to patients who have a 'false' cardiac arrest (i.e. a 'cardiac arrest team' call when the patient has

not had a cardiac arrest) may also improve outcome, because up to one third of these patients die during their in-hospital stay. Early recognition will also help to identify individuals for whom cardiorespiratory resuscitation is not appropriate or who do not wish to be resuscitated.

2.   Prevention of in-hospital cardiac arrest: the Chain of Prevention

The Chain of Prevention can assist hospitals in structuring care processes to prevent and detect patient deterioration and cardiac arrest. The five rings of the chain represent: staff education; the monitoring of patients; the recognition of patient deterioration; a system to call for help; and an effective response *(figure 3.1)*:

- **Education:** how to observe patients; interpretation of observed signs; the recognition of signs of deterioration; and the use of the ABCDE approach and simple skills to stabilise the patient pending the arrival of more experienced help.

- **Monitoring:** patient assessment and the measurement and recording of vital signs, which may include the use of electronic monitoring devices.

- **Recognition:** encompasses the tools available to identify patients in need of additional monitoring or intervention, including suitably designed vital signs charts and sets of predetermined 'calling criteria' to 'flag' the need to escalate monitoring or to call for more expert help.

- **Call for help:** protocols for summoning a response to a deteriorating patient should be universally known and understood, unambiguous and mandated. Doctors and nurses often find it difficult to ask for help or escalate treatment as they feel their clinical judgement may be criticised. Hospitals should ensure all staff are empowered to call for help. A structured communication tool such as SBAR (Situation, Background, Assessment, Recommendation) or RSVP (Reason, Story, Vital Signs, Plan) should be used to call for help.

- **Response:** to a deteriorating patient must be assured, of specified speed and by staff with appropriate acute or critical care skills, and experience.
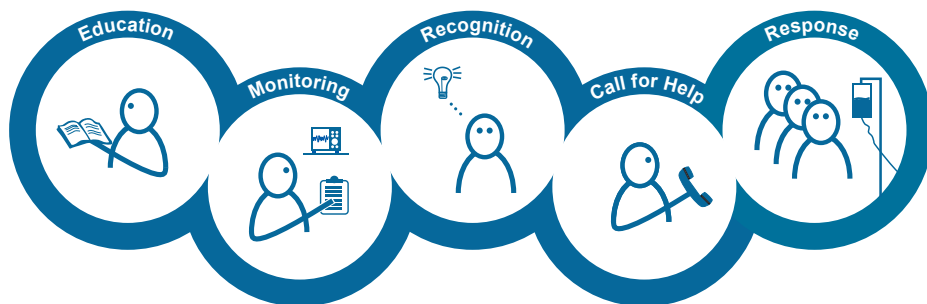
Figure 3.1
Chain of Prevention

Table 3.1

Example of early warning scoring (EWS) system - these values serve as general guidance and may vary in specific patient populations*

From Prytherch et al. ViEWS - Towards a national early warning score for detecting adult in-patient deterioration. Resuscitation. 2010;81(8):932-7

Score	3	2	1	0	1	2	3
Pulse (min^{-1})		≤ 40	41-50	51-90	91 - 110	111 - 130	≥ 131
Respiratory rate (min^{-1})	≤ 8		9-11	12-20		21-24	≥ 25
Temperature (°C)	≤ 35.0		35.1 - 36.0	36.1 - 38.0	38.1 - 39.0	≥ 39.1	
Systolic BP (mmHg)	≤ 90	91-100	101-110	111-249	≥ 250		
Oxygen saturation (%)	≤ 91	92-93	94-95	≥ 96			
Inspired oxygen				Air			Any oxygen therapy
AVPU				Alert (A)			Voice (V) Pain (P) Unresponsive (U)

3.   Recognising the deteriorating patient

In general, the clinical signs of critical illness are similar whatever the underlying process because they reflect failing respiratory, cardiovascular, and neurological systems i.e. ABCDE problems *(see below)*. Abnormal physiology is common on general wards, yet the measurement and recording of important physiological observations of acutely ill patients occurs less frequently than is desirable. The assessment of very simple vital signs, such as respiratory rate, may help to predict cardiorespiratory arrest. To help early detection of critical illness, many hospitals use early warning scores (EWS) or calling criteria. Early warning scoring systems allocate points to measurements of routine vital signs on the basis of their derangement from an arbitrarily agreed 'normal' range. The weighted score of one or more vital sign observations, or the total EWS, indicates the level of intervention required, e.g. increased frequency of vital signs monitoring, or calling ward doctors or resuscitation teams to the patient. An example of an EWS system is shown in table 3.1.

Early warning scores are dynamic and change over time and the frequency of observations should be increased to track improvement or deterioration in a patient's condition. If it is clear a patient is deteriorating help should be called for early rather than waiting for the patient to reach a specific score.

The patient's EWS is calculated based on table 3.1. An increased score indicates an increased risk of deterioration and death. There should be a graded response to scores according to local hospital protocols *(table 3.2)*.

Alternatively, systems incorporating calling criteria are based on routine observations, which activate a response when one or more variables reach an extremely abnormal value. It is not clear which of these two systems is better. Some hospitals combine elements of both systems.

Even when doctors are alerted to a patient's abnormal physiology, there is often delay in attending to the patient or referring to higher levels of care.

4. Response to critical illness

The traditional response to cardiac arrest is reactive: the name 'cardiac arrest team' implies that it will be called only after cardiac arrest has occurred. In some hospitals the cardiac arrest team has been replaced by other resuscitation teams (e.g. rapid response team, critical care outreach team, medical emergency team). These teams can be activated according to the patient's EWS (see above) or according to specific calling criteria. For example, the medical emergency team (MET) responds not only to patients in cardiac arrest, but also to those with acute physiological deterioration. The MET usually comprises medical and nursing staff from intensive care and general medicine and responds to specific calling criteria *(table 3.3)*.

Any member of the healthcare team can initiate a MET call. Early involvement of the MET may reduce cardiac arrests, deaths and unanticipated ICU admissions, and may facilitate decisions about limitation of treatment (e.g. do-not-attempt-resuscitation [DNAR] decisions). Medical emergency team interventions often involve simple tasks such as starting oxygen therapy and intravenous fluids. The benefits of the MET system remain to be proved.

Table 3.2
Example escalation protocol based on early warning score (EWS)

EWS	MINIMAL observation frequency	Escalation	
		Recorder's action	**Doctor's action**
3-5	4 hourly	Inform nurse in charge	
6	4 hourly	Inform doctor	Doctor to see within 1 hour
7-8	1 hourly	Inform doctor Consider continuous monitoring	Doctor to see within 30 minutes and discuss with senior doctor and/or outreach team
≥ 9	30 minutes	Inform doctor Start continuous monitoring	Doctor to see within 15 minutes and discuss with senior doctor and ICU team

Table 3.3
Medical emergency team (MET) calling criteria

MET calling criteria	
Airway	Threatened
Breathing	All respiratory arrests Respiratory rate < 5 min^{-1} Respiratory rate > 36 min^{-1}
Circulation	All cardiac arrests Pulse rate < 40 min^{-1} Pulse rate > 140 min^{-1} Systolic blood pressure < 90 mmHg
Neurology	Sudden decrease in level of consciousness Decrease in GCS of > 2 points Repeated or prolonged seizures
Other	Any patient causing concern who does not fit the above criteria

All critically ill patients should be admitted to an area that can provide the greatest supervision and the highest level of organ support and nursing care. This is usually in a critical care area, e.g. ICU, high dependency unit (HDU), or resuscitation room. These areas should be staffed by doctors and nurses experienced in advanced resuscitation and critical care skills.

Hospital staffing tends to be at its lowest during the night and at weekends. This influences patient monitoring, treatment and outcomes. Admission to general wards in the evening, or to hospital at weekends, is associated with increased mortality. Studies have shown that in-hospital cardiac arrests occurring in the late afternoon, at night or at weekends are more often non-witnessed and have a lower survival rate. Patients discharged at night from ICUs to general wards have an increased risk of ICU readmission and in-hospital death compared with those discharged during the day and those discharged to HDUs.

5. Causes of deterioration and cardiorespiratory arrest

Deterioration and cardiorespiratory arrest can be caused by primary airway and/or breathing and/or cardiovascular problems.

5.1. Airway obstruction

For a detailed review of airway management see chapter 7.

5.1.1. Causes

Airway obstruction can be complete or partial. Complete airway obstruction rapidly causes cardiac arrest. Partial obstruction often precedes complete obstruction. Partial airway obstruction can cause cerebral or pulmonary oedema, exhaustion, secondary apnoea, and hypoxic brain injury, and eventually cardiac arrest.

Causes of airway obstruction
• Central nervous system depression
• Blood
• Vomitus
• Foreign body (e.g. tooth, food)
• Direct trauma to face or throat
• Epiglottitis
• Pharyngeal swelling (e.g. infection, oedema)
• Laryngospasm
• Bronchospasm – causes narrowing of the small airways in the lung
• Bronchial secretions
• Blocked tracheostomy

Central nervous system depression may cause loss of airway patency and protective reflexes. Causes include head injury and intracerebral disease, hypercarbia, the depressant effect of metabolic disorders (e.g. diabetes mellitus), and drugs, including alcohol, opioids and general anaesthetic agents. Laryngospasm can occur with upper airway stimulation in a semi-conscious patient whose airway reflexes remain intact.

In some people, the upper airway can become obstructed when they sleep (obstructive sleep apnoea). This is more common in obese patients and obstruction can be worsened in the presence of other factors (e.g. sedative drugs).

5.1.2. Recognition

Assess the patency of the airway in anyone at risk of obstruction. A conscious patient will complain of difficulty in breathing, may be choking, and will be distressed. With partial airway obstruction, efforts at breathing will be noisy.

Complete airway obstruction is silent and there is no air movement at the patient's mouth. Any respiratory movements are usually strenuous. The accessory muscles of respiration will be involved, causing a 'see-saw' or 'rocking-horse' pattern of chest and abdominal movement: the chest is drawn in and the abdomen expands on inspiration, and the opposite occurs on expiration.

5.1.3. Treatment

The priority is to ensure that the airway remains patent.
Treat any problem that places the airway at risk; for example, suck blood and gastric contents from the airway and, unless contraindicated, turn the patient on their side.

Give oxygen as soon as possible to achieve an arterial blood oxygen saturation by pulse oximetry (SpO_2) in the range of 94-98 %. Assume actual or impending airway obstruction in anyone with a depressed level of consciousness, regardless of cause. Take steps to safeguard the airway and prevent further complications such as aspiration of gastric contents. This may involve nursing the patient on their side or with a head-up tilt.

Simple airway opening manoeuvres (head tilt-chin lift or jaw-thrust), insertion of an oropharyngeal or nasal airway, elective tracheal intubation or tracheostomy may be required. Consider insertion of a nasogastric tube to empty the stomach.

5.2. Breathing problems

5.2.1. Causes

Breathing inadequacy may be acute or chronic. It may be continuous or intermittent, and severe enough to cause apnoea (respiratory arrest), which will rapidly cause cardiac arrest. Respiratory arrest often occurs because of a combination of factors; for example, in a patient with chronic respiratory inadequacy, a chest infection, muscle weakness, or fractured ribs can lead to exhaustion, further depressing respiratory function. If breathing is insufficient to oxygenate the blood adequately (hypoxaemia), a cardiac arrest will occur eventually.

• **Respiratory drive**
Central nervous system depression may decrease or abolish respiratory drive. The causes are the same as those for airway obstruction from central nervous system depression.

• **Respiratory effort**
The main respiratory muscles are the diaphragm and intercostal muscles. The latter are innervated at the level of their respective ribs and may be paralysed by a spinal cord lesion above this level. The innervation of the diaphragm is at the level of the third, fourth and

fifth segment of the spinal cord. Spontaneous breathing cannot occur with severe cervical cord damage above this level.

Inadequate respiratory effort, caused by muscle weakness or nerve damage, occurs with many diseases (e.g. myasthenia gravis, Guillain-Barré syndrome, and multiple sclerosis). Chronic malnourishment and severe long-term illness may also contribute to generalised weakness.

Breathing can also be impaired with restrictive chest wall abnormalities such as kyphoscoliosis. Pain from fractured ribs or sternum will prevent deep breaths and coughing.

- **Lung disorders**

Lung function is impaired by a massive pleural effusion, a haemothorax, or pneumothorax. A tension pneumothorax causes a rapid failure of gas exchange, a reduction of venous return to the heart, and a fall in cardiac output. Severe lung disease will impair gas exchange. Causes include infection, aspiration, exacerbation of chronic obstructive pulmonary disease (COPD), asthma, pulmonary embolus, lung contusion, acute respiratory distress syndrome (ARDS) and pulmonary oedema.

5.2.2. Recognition

A conscious patient will complain of shortness of breath and be distressed. The history and examination will usually indicate the underlying cause. Hypoxaemia and hypercarbia can cause irritability, confusion, lethargy and a decrease in the level of consciousness. Cyanosis may be visible but is a late sign. A fast respiratory rate (> 25 min^{-1}) is a useful, simple indicator of breathing problems. Pulse oximetry is an easy, non-invasive measure of the adequacy of oxygenation *(see chapter 15)*.

However, it is not a reliable indicator of ventilation and an arterial blood gas sample is necessary to obtain values for arterial carbon dioxide tension ($PaCO_2$) and pH. A rising $PaCO_2$ and a decrease in pH are often late signs in a patient with severe respiratory problems.

5.2.3. Treatment

Give oxygen to all acutely ill hypoxaemic patients and treat the underlying cause. Give oxygen at 15 l min^{-1} using a high-concentration reservoir mask. Once the patient is stable, change the oxygen mask and aim for a SpO_2 in the range of 94-98 %. For example, suspect a tension pneumothorax from a history of chest trauma and confirm by clinical signs and symptoms. If diagnosed, decompress it immediately by inserting a large-bore (14 G) cannula into the second intercostal space, in the midclavicular line (needle thoracocentesis).

Patients who are having difficulty breathing or are becoming tired will need respiratory support. Non-invasive ventilation using a face mask or a helmet can be useful and prevent the need for tracheal intubation and ventilation. For patients who cannot breathe adequately, sedation, tracheal intubation and controlled ventilation are needed.

5.3. Circulation problems

5.3.1. Causes

Circulation problems may be caused by primary heart disease or by heart abnormalities secondary to other problems. Most often, circulation problems in acutely ill patients are due to hypovolaemia. The heart may stop suddenly or may produce an inadequate cardiac output for a period of time before stopping.

- **Primary heart problems**

The commonest cause of sudden cardiac arrest is an arrhythmia caused by either ischaemia or myocardial infarction. Cardiac arrest can also be caused by an arrhythmia due to other forms of heart disease, by heart block, electrocution and some drugs.

Sudden cardiac arrest may also occur with cardiac failure, cardiac tamponade, cardiac rupture, myocarditis and hypertrophic cardiomyopathy.

Causes of ventricular fibrillation
• Acute coronary syndromes *(chapter 4)*
• Hypertensive heart disease
• Valve disease
• Drugs (e.g. antiarrhythmic drugs, tricyclic antidepressants, digoxin)
• Inherited cardiac diseases (e.g. long QT syndromes)
• Acidosis
• Abnormal electrolyte concentration (e.g. potassium, magnesium, calcium)
• Hypothermia
• Electrocution

- **Secondary heart problems**

The heart is affected by changes elsewhere in the body. For example, cardiac arrest will occur rapidly following asphyxia from airway obstruction or apnoea, tension pneumothorax, or acute severe blood loss. Severe hypoxia and anaemia, hypothermia, oligaemia and severe septic shock will also impair cardiac function and this may lead to cardiac arrest.

5.3.2. Recognition

The signs and symptoms of cardiac disease include chest pain, shortness of breath, syncope, tachycardia, bradycardia, tachypnoea, hypotension, poor peripheral perfusion (prolonged capillary refill time), altered mental state, and oliguria.

Most sudden cardiac deaths (SCDs) occur in people with pre-existing cardiac disease, which may have been unrecognised previously. Although the risk is greater for patients with known severe cardiac disease, most SCDs occur in people with unrecognised disease. Asymptomatic or silent cardiac disease may include hypertensive heart disease, aortic valve disease, cardiomyopathy, myocarditis, and coronary disease.

- **Recognition of risk of sudden cardiac death out of hospital**

Coronary artery disease is the commonest cause of SCD. Non-ischaemic cardiomyopathy and valvular disease account for some other SCD events. A small percentage of SCDs are caused by inherited abnormalities (e.g. long and short QT syndromes, Brugada syndrome, hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy), and by congenital heart disease.

In patients with a known diagnosis of cardiac disease, syncope (with or without prodrome - particularly recent or recurrent) is as an independent risk factor for increased risk of death. Apparently healthy children and young adults who have SCD may also have symptoms and signs (e.g. syncope/pre-syncope, chest pain, palpitation, heart murmur) that should alert healthcare professionals to seek expert help to prevent cardiac arrest. Features that indicate a high probability of arrhythmic syncope include:

- syncope in the supine position

- syncope occurring during or after exercise (although syncope after exercise is often vasovagal)

- syncope with no or only brief prodromal symptoms

- repeated episodes of unexplained syncope

- syncope in individuals with a family history of sudden death or inherited cardiac condition

Assessment in a clinic specialising in the care of those at risk for SCD is recommended in family members of young victims of SCD or those with a known cardiac disorder resulting in an increased risk of SCD.

5.3.3. Treatment

Treat the underlying cause of circulatory failure. In many sick patients, this means giving intravenous fluids to treat hypovolaemia. Assess patients with chest pain for an acute coronary syndrome (ACS). A comprehensive description of the management of ACS is given in Chapter 4.

Most patients with cardiac ischaemic pain will be more comfortable sitting up. In some instances lying flat may provoke or worsen the pain. Consider using an antiemetic, especially if nausea is present.

Survivors of an episode of VF are likely to have a further episode unless preventative treatment is given. These patients may need percutaneous coronary intervention, coronary artery bypass grafting, or an implantable defibrillator.

Treating the underlying cause should prevent many secondary cardiac arrests; for example, early goal directed therapy to optimise vital organ perfusion decreases the risk of death in severe sepsis.

Cardiovascular support includes correction of underlying electrolyte or acid-base disturbances, and treatment to achieve a desirable cardiac rate, rhythm and output.

Advanced cardiovascular monitoring and echocardiography may be indicated. Appropriate manipulation of cardiac filling may require fluid therapy and vasoactive drugs. Inotropic drugs and vasoconstrictors may be indicated to support cardiac output and blood pressure. In some situations, mechanical circulatory support (e.g. intra-aortic balloon pump) or consideration of heart transplantation will be necessary.

6. The ABCDE approach

6.1. Underlying principles

The approach to all deteriorating or critically ill patients is the same. The underlying principles are:

1. Use the **A**irway, **B**reathing, **C**irculation, **D**isability, **E**xposure approach to assess and treat the patient.

2. Do a complete initial assessment and re-assess regularly.

3. Treat life-threatening problems before moving to the next part of assessment.

4. Assess the effects of treatment.

5. Recognise when you will need extra help. Call for appropriate help early.

6. Use all members of the team. This enables interventions, e.g. assessment, attaching monitors, intravenous access, to be undertaken simultaneously.

7. Communicate effectively - use the SBAR or RSVP approach *(see chapter 2)*.

8. The aim of the initial treatment is to keep the patient alive, and achieve some clinical improvement. This will buy time for further treatment and making a diagnosis.

9. Remember - it can take a few minutes for treatments to work.

6.2. First steps

1. Ensure personal and patient safety. Organize a safe setting and secure environment. Wear apron, gloves and glasses as appropriate.

2. First look at the patient in general to see if the patient appears unwell.

3. If the patient is awake, ask "How are you?". If the patient appears unconscious or has collapsed, shake him and ask "Are you alright?" If he responds normally he has a patent airway, is breathing and has brain perfusion. If he speaks only in short sentences, he may have breathing problems. Failure of the patient to respond is a clear marker of critical illness.

4. This first rapid "Look, Listen and Feel" of the patient should take about 30 seconds and will often indicate a patient is critically ill and there is a need for urgent help. Ask a colleague to ensure appropriate help is coming.

5. If the patient is unconscious, unresponsive, and is not breathing normally (occasional

gasps are not normal) start CPR according to the guidance in chapter 5. If you are confident and trained to do so, feel for a pulse to determine if the patient has a respiratory arrest. If there are any doubts about the presence of a pulse start CPR.

6. Monitor the vital signs early. Attach a pulse oximeter, ECG monitor and a non-invasive blood pressure monitor to all critically ill patients, as soon as possible.

7. Insert an intravenous cannula as soon as possible. Take bloods for investigation when inserting the intravenous cannula.

6.3. Airway (A)

Airway obstruction is an emergency. Get expert help immediately. Untreated, airway obstruction causes hypoxia and risks damage to the brain, kidneys and heart, cardiac arrest, and death.

1. Look for the signs of airway obstruction:

    -   Airway obstruction causes paradoxical chest and abdominal movements ('see-saw' respirations) and the use of the accessory muscles of respiration. Central cyanosis is a late sign of airway obstruction. In complete airway obstruction, there are no breath sounds at the mouth or nose. In partial obstruction, air entry is diminished and often noisy.

    -   In the critically ill patient, depressed consciousness often leads to airway obstruction.

2. Treat airway obstruction as a medical emergency:

    -   Obtain expert help immediately. Untreated, airway obstruction causes hypoxaemia (low PaO_2) with the risk of hypoxic injury to the brain, kidneys and heart, cardiac arrest, and even death.

    -   In most cases, only simple methods of airway clearance are required (e.g. airway opening manoeuvres, airways suction, insertion of an oropharyngeal or nasopharyngeal airway). Tracheal intubation may be required when these fail.

3. Give oxygen at high concentration:

    -   Provide high-concentration oxygen using a mask with an oxygen reservoir. Ensure that the oxygen flow is sufficient (usually 15 l min^{-1}) to prevent collapse of the reservoir during inspiration. If the patient's trachea is intubated, give high concentration oxygen with a self inflating bag.

    -   In acute respiratory failure, aim to maintain an oxygen saturation of 94-98 %. In patients at risk of hypercapnic respiratory failure *(see below)* aim for an oxygen saturation of 88-92 %.

6.4. Breathing (B)

During the immediate assessment of breathing, it is vital to diagnose and treat immediately life-threatening conditions, e.g. acute severe asthma, pulmonary oedema, tension pneumothorax, massive pleural effusion, rigidity of the thorax after severe burns to the chest, and massive haemothorax.

1. Look, listen and feel for the general signs of respiratory distress: sweating, central cyanosis, use of the accessory muscles of respiration, and abdominal breathing.

2. Count the respiratory rate. The normal rate is 12-20 breaths min^{-1}. A high (> 25 min^{-1}), or increasing, respiratory rate is a marker of illness and a warning that the patient may deteriorate suddenly.

3. Assess the depth of each breath, the pattern (rhythm) of respiration and whether chest expansion is equal on both sides.

4. Note any chest deformity (this may increase the risk of deterioration in the ability to breathe normally); look for a raised jugular venous pulse (JVP) (e.g. in acute severe asthma or a tension pneumothorax); note the presence and patency of any chest drains; remember that abdominal distension may limit diaphragmatic movement, thereby worsening respiratory distress.

5. Record the inspired oxygen concentration (%) and the SpO_2 reading of the pulse oximeter. The pulse oximeter does not detect hypercapnia. If the patient is receiving supplemental oxygen, the SpO_2 may be normal in the presence of a very high $PaCO_2$.

6. Listen to the patient's breath sounds a short distance from his face: rattling airway noises indicate the presence of airway secretions, usually caused by the inability of the patient to cough sufficiently or to take a deep breath. Stridor or wheeze suggests partial, but significant, airway obstruction.

7. Percuss the chest: hyper-resonance may suggest a pneumothorax; dullness usually indicates consolidation or pleural fluid.

8. Auscultate the chest: bronchial breathing indicates lung consolidation with patent airways; absent or reduced sounds suggest a pneumothorax or pleural fluid or lung consolidation caused by complete bronchial obstruction.

9. Check the position of the trachea in the suprasternal notch: deviation to one side indicates mediastinal shift (e.g. pneumothorax, lung fibrosis or pleural fluid).

10. Feel the chest wall to detect surgical emphysema or crepitus (suggesting a pneumothorax until proven otherwise).

11. The specific treatment of respiratory disorders depends upon the cause. Nevertheless, all critically ill patients should be given oxygen. In a subgroup of patients with chronic obstructive pulmonary disease (COPD), high concentrations of oxygen may depress breathing (i.e. they are at risk of hypercapnic respiratory failure - often referred to as type 2 respiratory failure). Nevertheless, these patients will also sustain end-organ damage or cardiac arrest if their blood oxygen tensions are allowed to decrease. In this group, aim for a lower than normal PaO_2 and oxygen

saturation. Give oxygen via a Venturi 28% mask (4 l min^{-1}) or a Venturi 24% mask (4 l min^{-1}) initially and reassess. Aim for target SpO$_2$ range of 88-92% in most COPD patients, but evaluate the target for each patient based on the patient's arterial blood gas measurements during previous exacerbations (if available). Some patients with chronic lung disease carry an oxygen alert card (that documents their target saturation) and their own appropriate Venturi mask.

12. If the patient's depth or rate of breathing is judged to be inadequate, or absent, use bag-mask or pocket mask ventilation to improve oxygenation and ventilation, whilst calling immediately for expert help.

   In cooperative patients who do not have airway obstruction consider the use of non-invasive ventilation (NIV). In patients with an acute exacerbation of COPD, the use of NIV is often helpful and prevents the need for tracheal intubation and invasive ventilation.

6.5.   Circulation (C)

In almost all medical and surgical emergencies, consider hypovolaemia to be the primary cause of shock, until proven otherwise. Unless there are obvious signs of a cardiac cause, give intravenous fluid to any patient with cool peripheries and a fast heart rate. In surgical patients, rapidly exclude haemorrhage (overt or hidden).

Remember that breathing problems, such as a tension pneumothorax, can also compromise a patient's circulatory state. This should have been treated earlier on in the assessment.

1. Look at the colour of the hands and digits: are they blue, pink, pale or mottled?

2. Assess the limb temperature by feeling the patient's hands: are they cool or warm?

3. Measure the capillary refill time (CRT). Apply cutaneous pressure for 5 s on a fingertip held at heart level (or just above) with enough pressure to cause blanching. Time how long it takes for the skin to return to the colour of the surrounding skin after releasing the pressure. The normal value for CRT is usually < 2 s. A prolonged CRT suggests poor peripheral perfusion. Other factors (e.g. cold surroundings, poor lighting, old age) can prolong CRT.

4. Assess the state of the veins: they may be under-filled or collapsed when hypovolaemia is present.

5. Count the patient's pulse rate (or preferably heart rate by listening to the heart with a stethoscope).

6. Palpate peripheral and central pulses, assessing for presence, rate, quality, regularity and equality. Barely palpable central pulses suggest a poor cardiac output, whilst a bounding pulse may indicate sepsis.

7. Measure the patient's blood pressure. Even in shock, the blood pressure may be normal, because compensatory mechanisms increase peripheral resistance in response to reduced cardiac output. A low diastolic blood pressure suggests arterial vasodilation (as in anaphylaxis or sepsis). A narrowed pulse pressure (difference

between systolic and diastolic pressures; normally 35-45 mmHg) suggests arterial vasoconstriction (cardiogenic shock or hypovolaemia) and may occur with rapid tachyarrhythmia.

8. Auscultate the heart. Is there a murmur or pericardial rub? Are the heart sounds difficult to hear? Does the audible heart rate correspond to the pulse rate?

9. Look for other signs of a poor cardiac output, such as reduced conscious level and, if the patient has a urinary catheter, oliguria (urine volume < 0.5 ml kg^{-1} h^{-1}).

10. Look thoroughly for external haemorrhage from wounds or drains or evidence of concealed haemorrhage (e.g. thoracic, intra-peritoneal, retroperitoneal or into gut). Intra-thoracic, intraabdominal or pelvic blood loss may be significant, even if drains are empty.

11. The specific treatment of cardiovascular collapse depends on the cause, but should be directed at fluid replacement, haemorrhage control and restoration of tissue perfusion. Seek the signs of conditions that are immediately life threatening, e.g. cardiac tamponade, massive or continuing haemorrhage, septicaemic shock, and treat them urgently.

12. Insert one or more large (14 or 16 G) intravenous cannulae. Use short, wide-bore cannulae, because they enable the highest flow.

13. Take blood from the cannula for blood gas analysis, routine haematological, biochemical, coagulation and microbiological investigations, and cross-matching, before infusing intravenous fluid.

14. If there is no suspected injury lift the legs of the patient or put the patient into the Trendelenburg position. If the heart rate decreases and the blood pressure improves give a rapid fluid challenge (over 5-10 min) of 500 ml of warmed crystalloid solution (e.g. Ringers lactate or 0.9 % sodium chloride) if the patient is normotensive. Give one litre, if the patient is hypotensive. Use smaller volumes (e.g. 250 ml) for patients with known cardiac failure or trauma and use closer monitoring (listen to the chest for crackles after each bolus).

15. Reassess the heart rate and BP regularly (every 5 min), aiming for the patient's normal BP or, if this is unknown, a target > 100 mmHg systolic.

16. If the patient does not improve, repeat the fluid challenge.

17. If symptoms and signs of cardiac failure (dyspnoea, increased heart rate, raised JVP, a third heart sound and pulmonary crackles on auscultation) occur, decrease the fluid infusion rate or stop the fluids altogether. Seek alternative means of improving tissue perfusion (e.g. inotropes or vasopressors).

18. If the patient has primary chest pain and a suspected ACS, record a 12-lead ECG early, and treat initially with aspirin, nitroglycerine, oxygen, and morphine. Treat ACS according to the guidance in chapter 4.

6.6. Disability (D)

Common causes of unconsciousness include profound hypoxia, hypercapnia, cerebral hypoperfusion, intoxication, or the recent administration of sedatives or analgesic drugs.

1. Review and treat the ABCs: exclude or treat hypoxia and hypotension.

2. Check the patient's drug chart for reversible drug induced causes of depressed consciousness. Give an antagonist where appropriate (e.g. naloxone for opioid toxicity).

3. Examine the pupils (size, equality and reaction to light).

4. Make a rapid initial assessment of the patient's conscious level using the AVPU method: **A**lert, responds to **V**ocal stimuli, responds to **P**ainful stimuli or **U**nresponsive to all stimuli. Alternatively, use the Glasgow Coma Scale score.

5. Measure the blood glucose to exclude hypoglycaemia using a rapid finger-prick bedside testing method. If the blood sugar is below 4.0 mmol l^{-1}, give an initial dose of 50 ml of 10 % glucose solution intravenously. If necessary, give further doses of intravenous 10 % glucose every minute until the patient has fully regained consciousness, or a total of 250 ml of 10 % glucose has been given. Repeat blood glucose measurements to monitor the effects of treatment. If there is no improvement consider further doses of 10 % glucose.

6. Consider other causes of reduced levels consciousness like electrolyte disorders or metabolic disorders (elevated plasma ammonia in patients with liver disease).

7. Nurse unconscious patients in the lateral position if their airway is not protected.

8. Recognise neurologic deficits e.g. aphasia and other signs of stroke.

6.7. Exposure (E)

To examine the patient properly full exposure of the body may be necessary. Respect the patient's dignity and minimise heat loss.

6.7.1. Additional information

1. Take a full clinical history from the patient, any relatives or friends, and other staff.

2. Review the patient's notes and charts:

    • Study both absolute and trended values of vital signs.

    • Check that important routine medications are prescribed and being given.

3. Review the results of laboratory or radiological investigations.

4. Consider which level of care is required by the patient (e.g. ward, HDU, ICU).

5. Make complete entries in the patient's notes of your findings, assessment and treatment. Where necessary, hand over the patient to your colleagues.

6.   Record the patient's response to therapy.

7.   Consider definitive treatment of the patient's underlying condition.

KEY LEARNING POINTS

• Most patients who have an in-hospital cardiac arrest have warning signs and symptoms before the arrest.

• Early recognition and treatment of the deteriorating patient will prevent some cardiorespiratory arrests.

• Use strategies such as early warning scoring (EWS) systems to identify patients at risk of cardiorespiratory arrest.

• Airway, breathing and circulation problems can cause cardiorespiratory arrest.

• Use the ABCDE approach to assess and treat critically ill patients.

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Chapter 4.

Acute coronary syndromes

> **LEARNING OUTCOMES**
>
> • the disease process which gives rise to acute coronary syndromes
>
> • how to differentiate between the acute coronary syndromes
>
> • the immediate treatment of acute coronary syndromes
>
> • management of patients after recovery from an acute coronary syndrome

1. Introduction

The acute coronary syndromes (ACS) comprise:

- unstable angina
- non-ST-segment-elevation myocardial infarction
- ST-segment-elevation myocardial infarction.

These clinical syndromes form parts of a spectrum of the same disease process. In the vast majority of cases this process is initiated by the fissuring of an atheromatous plaque in a coronary artery causing:

- haemorrhage into the plaque causing it to swell and restrict the lumen of the artery
- contraction of smooth muscle within the artery wall, causing further constriction of the lumen
- thrombus formation on the surface of the plaque, which may cause partial or complete obstruction of the lumen of the artery, or distal embolism

The extent to which these events reduce the flow of blood to the myocardium largely determines the nature of the clinical ACS that ensues.

1.1. Angina (stable and unstable)

Angina is pain or discomfort caused by myocardial ischaemia and is felt usually in or across the centre of the chest as tightness or an indigestion-like ache. As with acute myocardial

infarction (AMI), the pain/discomfort often radiates into the throat, into one or both arms, and into the back or into the epigastrium. Some patients experience angina predominantly in one or more of these areas rather than in the chest. Many patients perceive it as discomfort rather than pain. As with AMI, angina is sometimes accompanied by belching and this may be misinterpreted as evidence of indigestion as the cause of the discomfort. Pain of this nature, which is provoked only by exercise and which settles promptly when exercise ceases, is referred to as stable angina and is not an ACS.

In contrast, **unstable angina** is defined by one or more of:

1. Angina on exertion, occurring over a few days with increasing frequency, provoked by progressively less exertion. This is referred to as 'crescendo angina'.

2. Episodes of angina occurring recurrently and unpredictably, without specific provocation by exercise. These episodes may be relatively short-lived (e.g. a few minutes) and may settle spontaneously or be relieved temporarily by sublingual glyceryl trinitrate, before recurring within a few hours.

3. An unprovoked and prolonged episode of chest pain, raising suspicion of AMI, but without definite ECG or laboratory evidence of AMI *(see below)*.

    In unstable angina, the ECG may:

    a) be normal

    b) show evidence of acute myocardial ischaemia (usually horizontal or descending ST segment depression)

    c) show non-specific abnormalities (e.g. T wave inversion)

In unstable angina, cardiac enzymes are usually normal (but remember that there are causes other than myocardial infarction for elevated muscle enzymes such as creatin kinase [CK]), and troponin release is absent. ECG abnormality, especially ST segment depression, is a marker of increased risk of further coronary events in patients with unstable angina. However, a normal ECG and absent troponin release does not necessarily mean that a patient with unstable angina is not at high risk of early further life-threatening coronary events. Only if the ECG and troponin concentration are normal, and further risk assessment (e.g. by exercise testing or non-invasive imaging) does not indicate evidence of reversible myocardial ischaemia, should other possible causes of acute chest pain be considered if the initial history suggested unstable angina.

1.2. Non-ST-segment-elevation myocardial infarction (NSTEMI)

Acute myocardial infarction typically presents with chest pain that is felt as a heaviness or tightness or indigestion-like discomfort in the chest or upper abdomen, usually sustained for at least 20-30 min, often longer. The chest pain/discomfort often radiates into the throat, into one or both arms, into the back or into the epigastrium. Some patients experience the discomfort predominantly in one or more of these other areas rather than in the chest. Sometimes it may be accompanied by belching and this may be misinterpreted as evidence of indigestion as the cause of the discomfort.

When patients present with chest pain suggestive of AMI, non-specific ECG abnormalities such as horizontal or descending ST segment depression or T wave inversion (*figures 4.1 and 4.2)* or occasionally a normal ECG, and laboratory tests showing release of troponin (with or without elevated plasma concentrations of cardiac enzymes) this indicates that myocardial damage has occurred. This is referred to as NSTEMI. In this situation it is less likely that there has been abrupt complete occlusion of the culprit artery than in ST-segment-elevation MI (STEMI).

The amount of troponin or cardiac enzyme released reflects the extent of myocardial damage. Some of these patients will be at high risk of progression to coronary occlusion, more extensive myocardial damage, and sudden arrhythmic death. The risk of this is highest in the first few hours, days and months after the index event and diminishes progressively with time.

NSTEMI and unstable angina are classified together as 'non-ST-segment-elevation ACS' because the treatment of the two is essentially the same and differs in some respects from the treatment of STEMI. Treatment is dictated largely by assessment of risk.

1.3.   ST-segment-elevation myocardial infarction (STEMI)

A history of sustained acute chest pain typical of AMI, accompanied by acute ST segment elevation or new left bundle branch block (LBBB) on a 12-lead ECG is the basis for diagnosis of STEMI.

These findings almost always indicate on-going myocardial damage caused by acute complete occlusion of the 'culprit' coronary artery (after initial plaque fissuring). Left untreated there is likely to be further myocardial damage in the territory of the occluded artery, usually reflected in the development of Q waves and loss of R wave amplitude on the ECG. During the acute phase of STEMI there is a substantial risk of ventricular tachycardia (VT) and ventricular fibrillation (VF) and sudden death *(figure 4.3).*

2.   Diagnosis of acute coronary syndromes

The patient's history should be evaluated carefully during first contact with healthcare providers. It may provide the first clues for the presence of an ACS, trigger subsequent investigations and, in combination with information from other diagnostic tests, can help in making triage and therapeutic decisions in the out-of hospital setting and the emergency department (ED). Some patients (e.g. the elderly, diabetics, patients during the peri-operative period) may develop an ACS with little or no chest discomfort. The pain of angina or myocardial infarction is often mistaken for indigestion both by patients and healthcare professionals. Symptoms such as belching, nausea or vomiting are not helpful in distinguishing cardiac pain from indigestion; all may accompany angina and myocardial infarction.

2.1.   Clinical examination

Clinical examination is of limited benefit in the diagnosis of ACS. Severe pain of any source may provoke some of the clinical signs, such as sweating, pallor and tachycardia, which

commonly accompany ACS. History and examination are essential in order to recognise alternative, obvious causes for chest pain (e.g. localised severe chest wall tenderness), or to identify other life-threatening diagnoses (e.g. aortic dissection, pulmonary embolism). In aortic dissection the symptoms usually begin suddenly, and include severe chest pain. The pain may be described as sharp, stabbing, tearing, or ripping.

Examination may identify other important abnormalities (e.g. a cardiac murmur or signs of heart failure) that will influence choices of investigation and treatment. In patients with acute chest pain remember also to check for evidence of aortic dissection, especially if fibrinolytic therapy is intended. The presence of aortic dissection may be suggested by clinical signs such as loss of a pulse or asymmetry of the pulses in the upper limbs, acute aortic regurgitation, or signs of stroke from carotid artery involvement. Suspect aortic dissection in any patient whose acute chest pain is accompanied by marked hypotension but no obvious ECG evidence of AMI. However, in a patient with a good history and typical ECG evidence of STEMI do not delay reperfusion therapy without strong clinical evidence to justify prior investigation of possible aortic dissection.

Initial examination also serves as an important baseline so that changes, due either to progression of the underlying condition or in response to treatment, may be detected.

Also suspect extensive right ventricular (RV) infarction in patients with inferior or posterior STEMI who have elevated jugular venous pressure but no evidence of pulmonary oedema. Kussmaul's sign may be positive (JVP increases on inspiration). These patients are often hypotensive.

Figure 4.1
12-lead ECG showing acute ST-segment depression caused by myocardial ischaemia in a patient with a non-ST-segment ACS
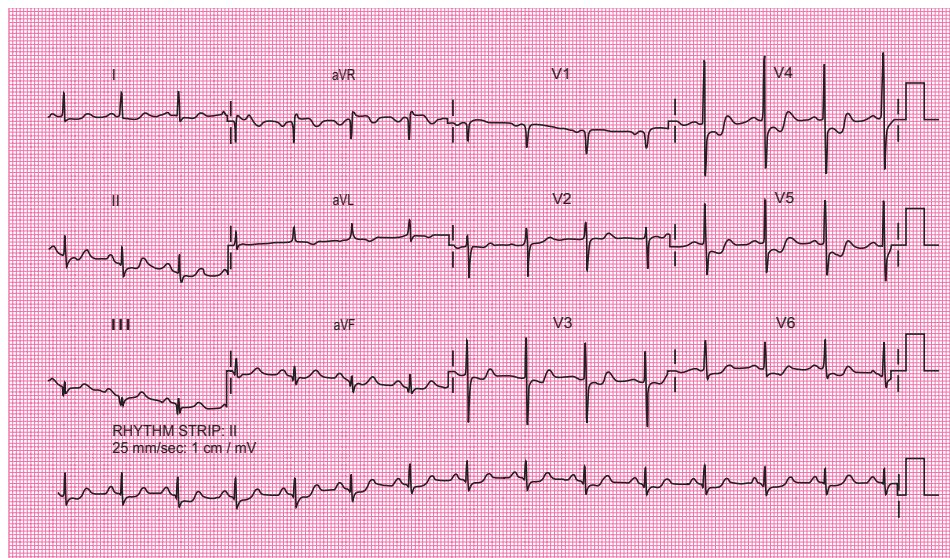
Figure 4.2

12-lead ECG showing T wave inversion in a patient with NSTEMI

4
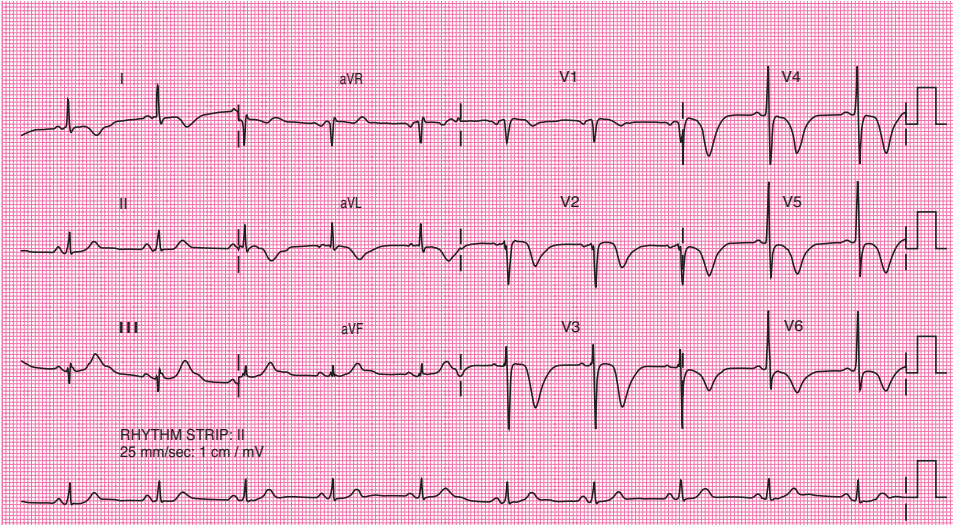
Figure 4.3

12-lead ECG showing onset of VF in a patient with an acute anteroseptal STEMI
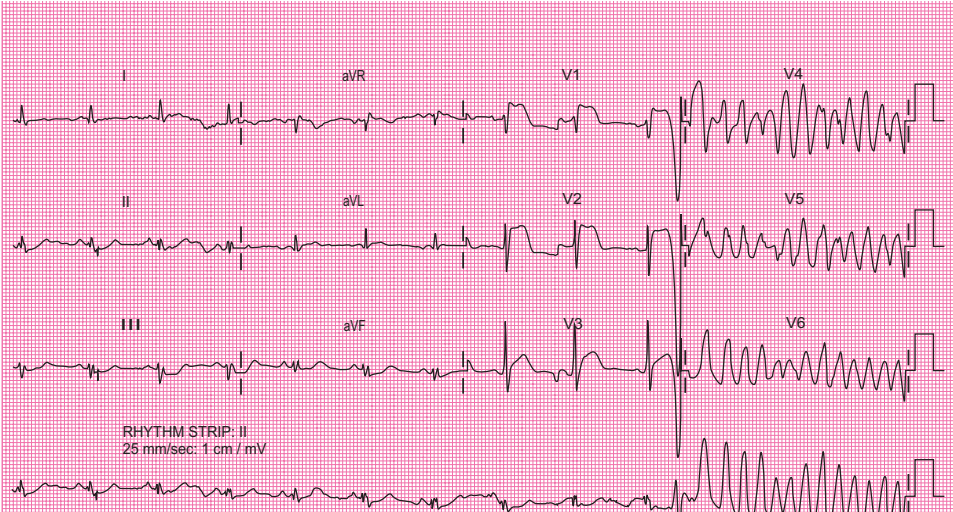
Figure 4.4

12-lead ECG showing an anterolateral STEMI
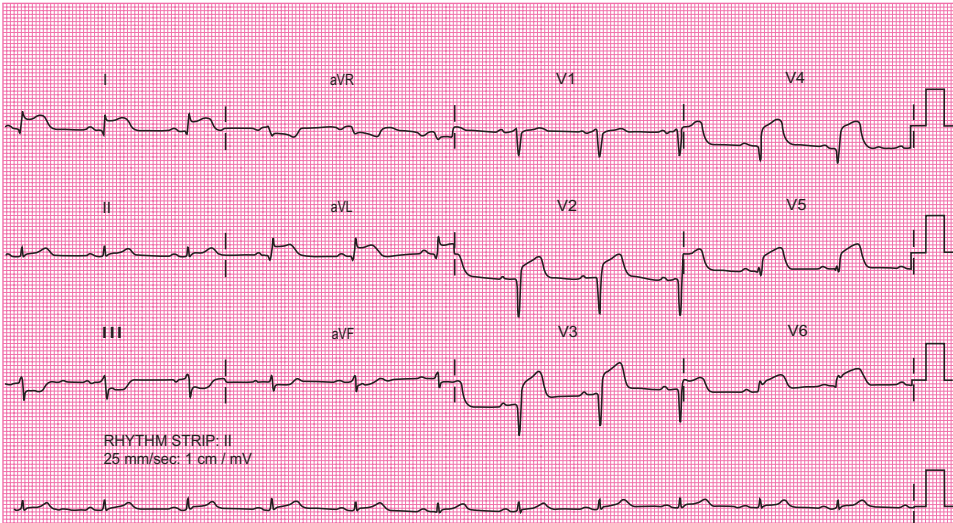
Figure 4.5

12-lead ECG showing an inferior STEMI
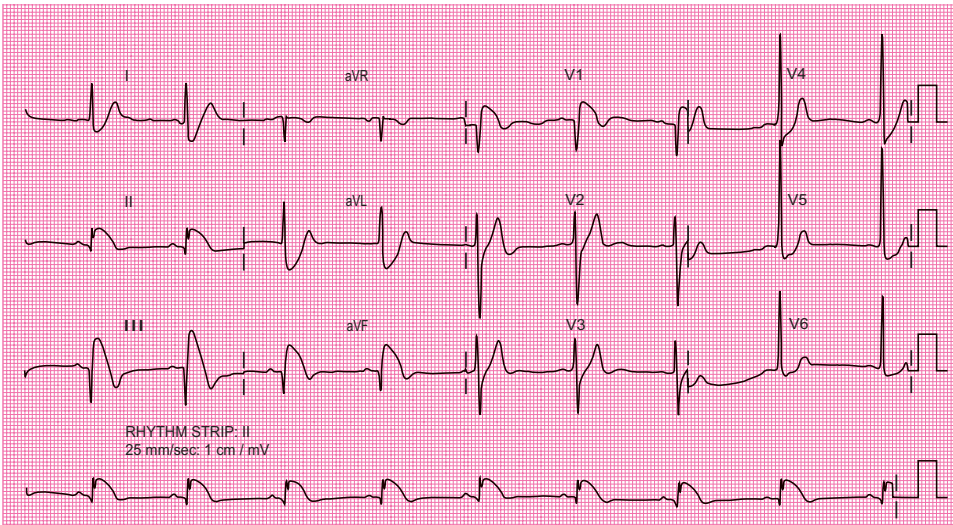
Figure 4.6
12-lead ECG showing a posterior STEMI
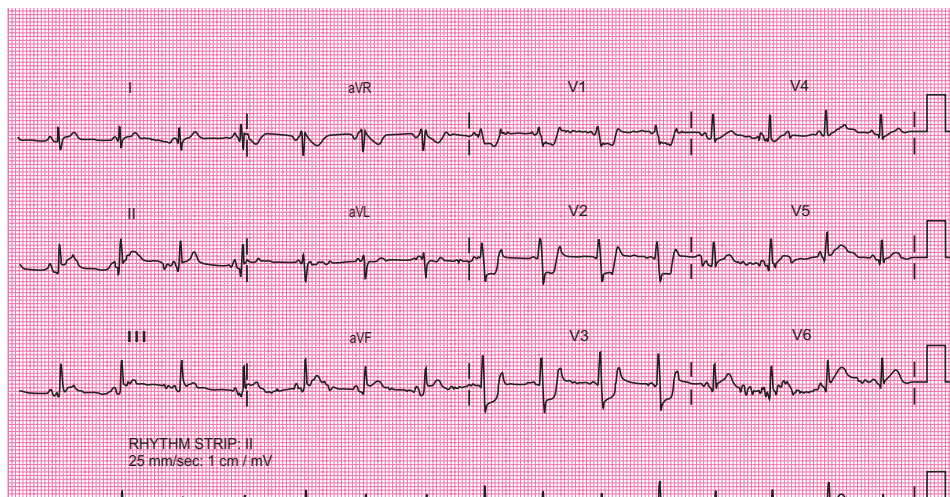
2.2. Investigations

2.2.1. The 12-lead ECG

A 12-lead ECG is the key investigation for assessment of an ACS. In the case of STEMI, it indicates the need for immediate reperfusion therapy (i.e. primary percutaneous coronary intervention (PCI) or pre-hospital fibrinolysis). When an ACS is suspected, printout of a 12-lead-ECG should be acquired and interpreted as soon as possible after first patient contact, to facilitate earlier diagnosis and triage. STEMI is typically diagnosed when, ST-segment elevation, measured at the J point, fulfills specific voltage criteria in the absence of left ventricular (LV) hypertrophy or left bundle branch block (LBBB). In patients with clinical suspicion of ongoing myocardial ischaemia with new or presumed new LBBB, consider prompt reperfusion therapy, preferably using primary PCI (PPCI). Ventricular pacing may also mask the presence of an evolving MI and may require urgent angiography to confirm diagnosis and initiate therapy.

Right precordial leads should be recorded in all patients with inferior STEMI in order to detect right ventricular MI. Isolated ST-depression ≥ 0.05 mV in leads V1 through V3 represents STEMI in the inferobasal portion of the heart which may be confirmed by ST segment elevation in posterior leads (V7-V9). Pre-hospital or ED ECG yields useful diagnostic information when interpreted by trained health care providers.

Recording of a 12-lead ECG out-of-hospital enables advanced notification to the receiving facility and expedites treatment decisions after hospital arrival. In many studies, using pre-hospital 12-lead ECG, the time from hospital admission to initiating reperfusion therapy

is reduced by 10 to 60 minutes. This is associated with shorter times to reperfusion and improved patient survival in both patients with PCI and those undergoing fibrinolysis.

Trained EMS personnel (emergency physicians, paramedics and nurses) can identify STEMI, defined by ST elevation of ≥ 0.1mV elevation in at least two adjacent limb leads or > 0.2mV in two adjacent precordial leads, with a high specificity and sensitivity comparable to diagnostic accuracy in the hospital. It is thus reasonable that paramedics and nurses be trained to diagnose STEMI without direct medical consultation, as long as there is strict concurrent provision of quality assurance.

If interpretation of the pre-hospital ECG is not available on-site, computer interpretation or field transmission of the ECG is reasonable. Recording and transmission of diagnostic quality ECGs to the hospital usually takes less than 5 minutes. When used for the evaluation of patients with suspected ACS, computer interpretation of the ECG may increase the specificity of diagnosis of STEMI, especially for clinicians inexperienced in reading ECGs. The benefit of computer interpretation; however, is dependent on the accuracy of the ECG report. Incorrect reports may mislead inexperienced ECG readers. Thus computer-assisted ECG interpretation should not replace, but may be used as an adjunct to, interpretation by an experienced clinician.

2.2.2. Biomarkers

In the absence of ST elevation on the ECG, the presence of a suggestive history and elevated concentrations of biomarkers (troponins, CK and CKMB) characterise non-STEMI and distinguish it from STEMI and unstable angina respectively. Measurement of a cardiac-specific troponin is used routinely because of its higher sensitivity and specificity. Elevated concentrations of troponin are particularly helpful in identifying patients at increased risk of adverse outcome.

In order to use the measured biomarker optimally, clinicians should be familiar with the sensitivity, precision and institutional norms of the assay, and also the release kinetics and clearance. Highly sensitive (ultrasensitive) cardiac troponin assays have been developed. They can increase sensitivity and accelerate diagnosis of MI in patients with symptoms suspicious of cardiac ischaemia.

Cardiac biomarker testing should be part of the initial evaluation of all patients presenting to the ED with symptoms suggestive of cardiac ischaemia. However, the delay in release of biomarkers from damaged myocardium prevents their use in diagnosing myocardial infarction in the first hours after the onset of symptoms. For patients who present within 6 hours of symptom onset, and have an initial negative cardiac troponin, biomarkers should be measured again between 2-3 and up to 6 hours later for hs-cTn (12 hours with regular troponin). The majority of patients with possible ACS do not have an ACS but the identification of those with ACS is challenging. The recently reported rate of patients with a 'missed' diagnosis of ACS in the ED is up to 3.5 % with significant morbidity and mortality.

- **Cardiac troponins (troponin T and troponin I)**
Cardiac-specific troponins are components of the contractile structure of myocardial cells. Because concentrations of troponin in the blood of healthy individuals are undetectably

low, and cardiac-specific troponins measured by current assays do not arise from extra-cardiac sources, the troponins are very sensitive and specific markers of cardiac injury. In the context of a typical clinical presentation of ACS, troponin release provides evidence of myocardial damage and therefore indicates myocardial infarction rather than unstable angina. In addition troponin measurement provides useful assessment of risk. The greater the troponin concentration, the greater is the risk of a further event. A combination of ST segment depression on the ECG and raised troponin identifies a particularly high-risk group for subsequent MI and sudden cardiac death.

It is not recommended to use high sensitivity cardiac troponins as a stand-alone measure at 0 and 2 hours to exclude the diagnosis of ACS, defined as < 1 % 30-day major adverse cardiovascular effects (MACE). Negative hs-cTnI measured at 0 and 2 hours may be used together with low risk stratification (TIMI score of 0 or 1) to exclude the diagnosis of ACS. Also negative cTnI or cTnT measured at 0 and 3-6 hours together may be used in conjunction with very low risk stratification (Vancouver score of 0 or North American CP score of 0 and age < 50) to exclude the diagnosis of ACS.

There is no evidence to support the use of troponin point-of-care testing (POCT) in isolation as a primary test in the pre-hospital setting to evaluate patients with symptoms suspicious of cardiac ischaemia. In the ED, use of point-of-care troponin assays may help to shorten time to treatment and length of ED stay.

2.2.3. Imaging techniques

Non invasive imaging techniques (CT angiography, cardiac magnetic resonance, myocardial perfusion imaging, and echocardiography) have been evaluated as means of screening these low-risk patients and identifying subgroups that can be discharged home safely.

Furthermore, differential diagnoses such as aortic dissection, pulmonary embolism, aortic stenosis, hypertrophic cardiomyopathy, pericardial effusion, or pneumothorax may be identified. Therefore, echocardiography should be routinely available in ED, and used in all patients with suspected ACS. Studies are needed to evaluate the impact of echocardiography in the pre-hospital setting.

Multi-detector computer tomography coronary angiography (MDCTCA). MDCTCA has been recently proposed in the management acute chest pain in the ED. It is accurate compared with invasive coronary angiography, enabling differential diagnosis, and it is feasible and practical in the ED. MDCTCA has a high ability to rule out obstructive coronary artery disease.

3.   Risk assessment

The choice of treatment is determined largely by the extent of myocardial damage and by the risk of early further coronary events.
Risk assessment scores and clinical prediction algorithms using clinical history, physical examination, ECG, and cardiac troponins have been developed to help identify patients with ACS at increased risk of adverse outcome(s).

Global Registry of Acute Coronary Events (GRACE) and Thrombolysis In Myocardial Infarction (TIMI) risk score are the most commonly used. In a recent meta-analysis, TIMI and GRACE risk scores were the only ones validated in multiple clinical setting, with GRACE showing a better performance.

In patients suspected of an ACS the combination of an unremarkable past history and physical examination with negative initial ECG and biomarkers cannot be used to exclude ACS reliably. Therefore a follow up period is mandatory in order to reach a diagnosis and make therapeutic decisions

4. Immediate treatment

4.1. General measures in all acute coronary syndromes

Start with a rapid clinical assessment and record at least one twelve lead ECG. Give immediate treatment to relieve symptoms, limit myocardial damage and reduce the risk of cardiac arrest. Immediate general treatment for ACS comprises:

- aspirin 300 mg, orally, crushed, chewed, or IV as soon as possible

- nitroglycerine, as sublingual glyceryl trinitrate (tablet or spray) unless patient is hypotensive or extensive RV infarction is suspected

- relief of pain is of paramount importance and intravenous morphine (or diamorphine) should be given, titrated to control symptoms but avoiding sedation and respiratory depression

- consider antithrombotic treatment with unfractioned (UFH) or low molecular weight heparin (LMWH) or fondaparinux

Most patients with cardiac ischaemic pain will be more comfortable sitting up. In some instances lying flat may provoke or worsen the pain. Give an anti-emetic with opiate analgesia or if the patient has nausea.

4.2. Treatment of STEMI (or AMI with new LBBB)

For patients presenting with STEMI within 12 h of symptom onset, mechanical or pharmacological reperfusion must be achieved without delay. The aim is to restore the blood supply to myocardium that has not yet been damaged irreversibly. Clinical trials have confirmed the effectiveness of reperfusion therapy in reducing infarct size, complications, and mortality from MI. Reperfusion therapy is most effective when undertaken early after the onset of myocardial infarction and the benefit diminishes progressively with delay.

The risk/benefit ratio for reperfusion therapy favours reperfusion therapy for those patients who are at highest risk of immediate major myocardial damage and death.

Beyond 12 h from the onset of chest pain, the risks of fibrinolytic therapy probably outweigh any small residual benefit, but emergency percutaneous coronary intervention (PCI) should be considered in this situation if there is ongoing clinical or ECG evidence of ischaemia.

4.3. Oxygen

Patients with acute chest pain with presumed ACS do not need supplemental oxygen unless they present with signs of hypoxia, dyspnoea or heart failure. There is increasing evidence suggesting that hyperoxia may be harmful in patients with uncomplicated myocardial infarction. In ACS complicated with cardiac arrest, hypoxia develops rapidly. Ischaemic brain injury is a major determinant for neurologically intact survival. Therefore during CPR adequate oxygenation is essential. After ROSC, avoid both hypoxia and hyperoxia (see post resuscitation care). Use 100 % inspired oxygen until the arterial oxygen saturation can be measured reliably. As soon as the arterial blood oxygen saturation can be measured reliably, titrate the inspired oxygen concentration to achieve arterial blood oxygen saturation in the range of 94-98 %.

4.4. Coronary reperfusion therapy

In STEMI, coronary reperfusion may be achieved in one of two ways:

- Percutaneous coronary intervention (PCI) may be used to re-open the occluded artery. This is referred to as primary PCI.

- Fibrinolytic therapy may be given in an attempt to dissolve the occluding thrombus that precipitated the MI.

Coronary angioplasty with or without stent placement has become the first-line treatment for patients with STEMI. PPCI performed with a limited delay to first balloon inflation after first medical contact, at a high-volume centre, by an experienced operator who maintains an appropriate expert status, is the preferred treatment as it improves morbidity and mortality as compared with immediate fibrinolysis. Coronary angiography is used to identify the occluded coronary artery, following which a guidewire is passed through the occluding thrombus, enabling a deflated balloon to be positioned at the site of occlusion and inflated to re-open the artery. Aspiration devices may be used to remove thrombus from the vessel and glycoprotein IIb/IIIa inhibitors may be injected intravenously or directly into the coronary artery. It is usual practice to insert a stent into the segment of previously occluded artery, to reduce the risk of re-occlusion at this point.

4.4.1. Primary PCI

Primary PCI is the most reliable method of re-opening of the culprit artery in the majority of patients. Coronary artery patency can be confirmed, secured and maintained. There is a lower risk of major, particularly intracranial, bleeding than with fibrinolytic therapy.

For PPCI, to provide reliable timely reperfusion, a fully-equipped catheter laboratory staffed by an experienced team must be available 24 hours a day. A fail-safe pathway of communication and care must be implemented in each region in order that patients in whom STEMI is diagnosed can access the service, ideally by direct transfer to this facility. Primary PCI can then be offered to patients for whom a 'first medical contact-to-balloon' time of 120 min can be achieved (ESC Guidelines 2010). In patients who present within

2 hours of onset of chest pain the time from first medical contact to reperfusion should be less than 90 min. Longer delays are associated with higher mortality.

Where PPCI is not available immediately, the need to achieve reperfusion as early as possible remains a high priority and for those patients initial treatment by fibrinolytic therapy may offer the best chance of achieving early reperfusion.

Time delay to PPCI may be significantly shortened by improving the systems of care:

- A pre-hospital ECG should be acquired as soon as possible and interpreted for the diagnosis of STEMI. This can reduce mortality in both patients planned for PPCI and fibrinolytic therapy.

- STEMI recognition may be accomplished by ECG transmission or onsite interpretation by physicians, or highly trained nurses or paramedics, with or without the aid of computer ECG interpretation.

- When PPCI is the planned strategy, pre-hospital activation of catheterisation laboratory for PPCI will contribute to a mortality benefit.

Additional elements for an effective system of care include:

- Requiring the catheterisation laboratory to be ready within 20 minutes available 24/7.

- Providing real-time data feedback on the real time course from symptom onset to PCI.

An injectable anticoagulant must be used in primary PCI for STEMI.

- unfractionated Heparin > 5000 IU
- Enoxaparin

4.4.2. Fibrinolytic therapy

Fibrinolytic therapy has been shown in large-scale clinical trials to provide substantial reduction in mortality from AMI when given during the first few hours after the onset of chest pain. One of the major advantages of fibrinolytic therapy is that it does not require a cardiac catheter laboratory or an operator skilled in angioplasty. Early reperfusion may be achieved by pre-hospital fibrinolytic therapy with resulting clinical benefit, particularly when transport times to hospital are very long. Early treatment may also be achieved by minimising door-to-needle time (time from arrival at hospital to administration of fibrinolytic therapy).

Table 4.1
Typical indications for immediate reperfusion therapy for AMI

Typical indications for immediate reperfusion therapy for AMI
Presentation within 12 hours of onset of chest pain suggestive of AMI and:
ST segment elevation > 0,2 mV in 2 adjacent chest leads, or > 0,1 mV in 2 or more 'adjacent' limb leads; or
Dominant R waves and ST depression in V1-V3 (posterior infarction); or
New-onset (or presumed new-onset) LBBB.

4

Table 4.2
Typical contraindications to fibrinolytic therapy

Typical contraindications to fibrinolytic therapy
ABSOLUTE
• Previous haemorrhagic stroke
• Ischaemic stroke during the previous 6 months
• Central nervous system damage or neoplasm
• Recent (within 3 weeks) major surgery, head injury or other major trauma
• Active internal bleeding (menses excluded) or gastro-intestinal bleeding within the past month
• Known or suspected aortic dissection
• Known bleeding disorder
RELATIVE
• Refractory hypertension (systolic blood pressure > 180 mmHg)
• Transient ischaemic attack in preceding 6 months
• Oral anticoagulant treatment
• Pregnancy or less than 1 week post-partum
• Non-compressible vascular puncture
• Active peptic ulcer disease
• Advanced liver disease
• Infective endocarditis
• Previous allergic reaction to the fibrinolytic drug to be used

Fibrinolytic therapy carries a risk of bleeding, including cerebral haemorrhage, and not all patients can be given this treatment safely. Table 4.1 lists typical indications for reperfusion therapy and the typical contraindications to fibrinolytics are shown in table 4.2. Most of these contra-indications are relative; the experienced clinician will decide whether the benefit from fibrinolytic therapy outweighs the risk to the individual patient or whether emergency angiography with a view to primary PCI would be more appropriate.

Figure 4.7 describes the options for reperfusion therapy for STEMI in the form of an algorithm.

4.4.3. Platelet inhibition and anticoagulant therapy with fibrinolytic therapy

Give all patients receiving a fibrinolytic agent for STEMI:

- aspirin 300 mg, and

- clopidogrel as a 600 mg loading dose, and

- antithrombin therapy: low molecular weight heparin (IV bolus then SC) or unfractionated heparin (full dose) or fondaparinux.

4.5. Rescue angioplasty

In 20-30 % of patients receiving a fibrinolytic for STEMI, reperfusion is not achieved. Observe patients closely with cardiac monitoring during and after administration of a fibrinolytic. Record a 12-lead ECG at 60-90 min after giving fibrinolytic therapy. Failure of ST segment elevation to resolve by more than 50 % compared with the pre-treatment ECG suggests that fibrinolytic therapy has failed to re-open the culprit artery. Symptoms are a less accurate guide to reperfusion because most patients will have received opiate analgesia. Even after initially successful thrombolysis there is a significant risk of re-occlusion and patients should be admitted to a coronary care unit with continuous ECG monitoring.

In cases of failed reperfusion or re-occlusion/re-infarction transfer the patient immediately to a cardiac catheter laboratory for mechanical reperfusion (PCI). In failed thrombolysis this is referred to as rescue PCI and has been shown to improve event-free survival and reduce heart failure when compared to conservative therapy or repeat fibrinolytic therapy. Again rescue PCI must be performed without any time-delay in order to be effective.

The role of 'facilitated PCI' in which initial fibrinolytic therapy is followed by immediate coronary angiography and PCI remains a subject of ongoing debate. So far there is insufficient evidence in support of this strategy but trials are ongoing.

4.6. Inhibitors of platelet aggregation

Platelet activation and aggregation following atherosclerotic plaque rupture are central pathophysiologic mechanisms of acute coronary syndromes and antiplatelet therapy is a pivotal treatment of ACS whether with or without ST segment elevation, with or without reperfusion and with or without revascularisation.

- **Acetylsalicylic acid (ASA)**

Give an oral loading dose of ASA (150 to 300 mg of a non-enteric coated formulation) or 150 mg of an IV preparation as soon as possible to all patients with suspected ACS unless the patient has a known true allergy to ASA or has active bleeding. ASA may be given by the first healthcare provider, bystander or by dispatcher assistance according to local protocols.

- **ADP Receptor inhibitors**

The inhibition of the platelet ADP receptor by the thienopyridines clopidogrel and prasugrel (irreversible inhibition) and the cyclo-pentyl-triazolo-pyrimidine ticagrelor (reversible inhibition) leads to further platelet aggregation inhibition, in addition to that produced by ASA. In contrast to clopidogrel, the effect of prasugrel and ticagrelor are largely independent of a genetically determined variability of drug metabolism and activation. Therefore prasugrel and ticagrelor (reversible) lead to a more reliable, faster and stronger inhibition of platelet aggregation.

4.6.1.   ADP-receptor inhibitors in non-STEMI-ACS

- **Clopidogrel**

If given in addition to heparin and ASA in high-risk non-STEMI-ACS patients, clopidogrel improves outcome.[83] If a conservative approach is selected, give a loading dose of 300 mg; with a planned PCI strategy, an initial dose of 600 mg may be preferred. There is no large-scale study investigating pre-treatment with clopidogrel, compared with peri-interventional application – either with a 300 mg or 600 mg loading dose.

- **Prasugrel**

Prasugrel (60 mg loading dose) may be given to patients with high-risk non-STEMI-ACS and planned PCI only after angiography, provided that coronary stenoses are suitable for PCI. Contraindications (history of TIA/stroke) and the benefit - risk balance in patients with high bleeding risk (weight < 60 kg, age > 75 years) should be considered.

- **Ticagrelor**

According to the latest ESC guidelines, ticagrelor (180 mg loading dose) should be given in addition to ASA in all patients with moderate to high-risk non-STEMI-ACS whether an invasive strategy is planned or not. In patients with non-STEMI-ACS planned for a conservative approach, give ticagrelor or clopidogrel as soon as the diagnosis is confirmed. There is insufficient evidence to recommend for or against pre-treatment with these agents when PCI is the initial strategy.

4.7.   Treatment of unstable angina and NSTEMI

Parenteral anticoagulation, in addition to anti-platelet drugs, is recommended at the time of diagnosis because it effectively reduces the rate of major cardiovascular events (MACE) in patients with non-STEMI-ACS.

Figure 4.7
Access to reperfusion therapy for STEMI

* In patients presenting < 2 h after onset of pain, time from first medical contact to PCI should be less than 90 min. If not achievable consider immediate fibrinolytic therapy.
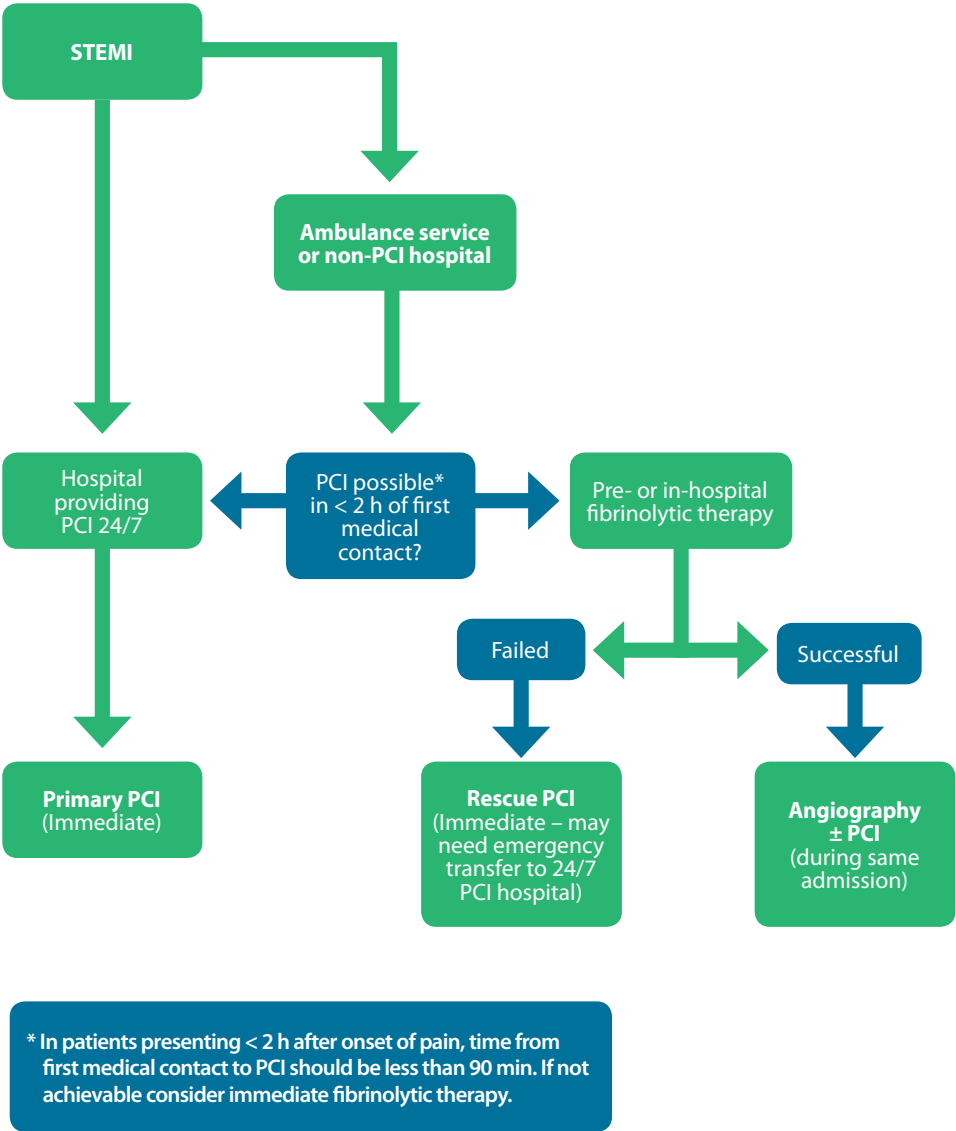
5. Subsequent management of patients with acute coronary syndromes

5.1. Suspected unstable angina – low risk patients

Patients with suspected unstable angina without a definite history of preceding angina of effort or myocardial infarction and without high-risk features at presentation (ECG and troponin levels normal after 6-8 h) should undergo early further risk assessment either by exercise testing or non-invasive imaging.

5.2. Suspected angina – high risk unstable angina and NSTEMI

Patients with unstable angina and high-risk features (e.g. resting ST segment depression, high-risk features on exercise test or non-invasive imaging) should be considered for early investigation by invasive coronary angiography.

Patients with NSTEMI should be regarded as a high-risk group, requiring early assessment by coronary angiography during the same hospital admission in the majority of cases, ideally within 72 h.

Many patients in both these groups will benefit from revascularisation by PCI. A few may require coronary artery bypass grafts (CABG).

Formal risk-scoring systems such as GRACE (Global Registry of Acute Coronary Events) should be used to guide clinical management. Those patients at the highest risk derive the greatest benefit from early intervention in terms of reducing further major cardiac events.

5.3. STEMI

If fibrinolytic therapy has been used, many patients will be left with a severe stenosis or unstable plaque in the culprit coronary artery. PCI can stabilise this situation and reduce the risk of re-occlusion of the artery and resulting further myocardial infarction, cardiac arrest or sudden death. Coronary angiography and, if indicated, PCI should be undertaken early during the same hospital admission.

In patients with completed STEMI who have not been treated with reperfusion therapy (e.g. because of late presentation) it is usually recommended that coronary angiography is undertaken during the same hospital admission. Although the benefits of re-opening an occluded culprit artery late after STEMI are uncertain, there is often disease in other coronary vessels that can give rise to further major coronary events over subsequent months. Defining the severity and anatomy of such disease can help to identify those at highest risk, in whom early intervention may reduce that risk.

5.4. Ventricular arrhythmia complicating acute coronary syndromes

When ventricular arrhythmia complicates an acute coronary syndrome, interpret its significance in the context of the precise clinical setting and the time of onset of the arrhythmia. When VF/pVT cardiac arrest occurs within the first 24-48 h after STEMI, and subsequent recovery is uncomplicated, the risk of another ventricular arrhythmia is relatively low and is determined by other factors, especially the severity of left ventricular impairment. If VF/pVT cardiac arrest occurs in the context of non-ST segment elevation ACS, there may be a continuing risk of further ventricular arrhythmia. If the arrhythmia has been caused by severe myocardial ischaemia, very urgent revascularisation is needed to prevent recurrence of the ischaemia and reduce the risk of resulting arrhythmia. If this is not achievable or if the arrhythmia has occurred without evidence of severe ischaemia, the patient will be at risk of recurrent ventricular arrhythmia and should be referred to a heart rhythm specialist with a view to insertion of an implantable cardioverter-defibrillator (ICD) **before discharge from hospital.**

Patients who have a VF/pVT cardiac arrest as a late complication after myocardial infarction, or outside the context of an ACS, will be at risk of recurrent cardiac arrest and should be seen urgently by a heart rhythm specialist with a view to ICD implantation **before discharge from hospital.**

5.4.1. Reperfusion after successful CPR

As it is often accompanied by an acute coronary artery occlusion or by a high degree stenosis, acute coronary syndrome (ACS) is a frequent cause of out-of-hospital cardiac arrest (OHCA) The invasive management (i.e. early coronary angiography (CAG) followed by immediate PCI if deemed necessary) of this patient group, particularly patients after prolonged resuscitation and having nonspecific ECG changes, has been controversial due to the lack of specific evidence and significant implications on resource utilization (including transfer of patients to PCI centres).

- **PCI Following ROSC with ST-Elevation**
The highest prevalence of acute coronary lesion is observed in patients with ST segment elevation (STE) or left bundle branch block (LBBB) on post-ROSC electrocardiogram (ECG). It is highly probable that early invasive management is a strategy associated with a clinically relevant benefit in these patients

Based on the available data, emergent cardiac catheterization lab evaluation (and immediate PCI if required) should be performed in selected adult patients with ROSC after OHCA of suspected cardiac origin with ST segment elevation on ECG.

Observational studies also indicate that optimal outcomes after OHCA are achieved with a combination of targeted temperature management and PCI, which can be combined in a standardized post–cardiac-arrest protocol as part of an overall strategy to improve neurologically intact survival in this patient group.

- **PCI Following ROSC Without ST-Elevation**

In contrast to the usual presentation of ACS in non cardiac arrest patients, recommended tools to assess coronary ischaemia are less accurate in this setting. It is reasonable to discuss an emergent cardiac catheterization lab evaluation after ROSC in patients with the highest risk of coronary cause of CA. A variety of factors such as patient age, duration of CPR, haemodynamic instability, presenting cardiac rhythm, neurologic status upon hospital arrival, and perceived likelihood of cardiac aetiology can influence the decision to undertake the intervention. A recent consensus statement from the European Association for Percutaneous Cardiovascular Interventions (EAPCI) has emphasised that in OHCA patients, cardiac catheterisation should be performed immediately in the presence of ST-elevation and considered as soon as possible (less than two hours) in other patients in the absence of an obvious non-coronary cause, particularly if they are haemodynamically unstable. In patients who present in a non-PCI centre transfer for angiography and PPCI if indicated should be considered on an individual basis, weighing the expected benefits from early angiography against the risks from patient transport.

5.5. Other complications of ACS

5.5.1. Heart failure

Patients with heart failure complicating AMI or other ACS are at increased risk of deterioration, cardiac arrest and death: prompt, effective treatment of the heart failure is required to reduce risk. Give a loop diuretic (e.g. furosemide) and/or glyceryl trinitrate (sublingual and/or intravenous) for immediate symptomatic treatment. Give regular loop diuretic to maintain symptom control but review the need for this and the dose at least daily for the first few days. Ensure that angiotensin converting enzyme inhibitor (ACEI) treatment has been started and increase the dose as tolerated, until the target dose is achieved. In patients intolerant of ACEI, consider an angiotensin receptor blocker. Maintain beta blockade unless contraindicated or not tolerated. If more than mild LV systolic impairment is confirmed (ejection fraction 40 % or less) consider addition of an aldosterone antagonist (e.g. eplerenone or spironolactone).

5.5.2. Cardiogenic shock

This consists of severe hypotension with poor peripheral perfusion, often accompanied by pulmonary oedema, drowsiness or mental confusion due to poor cerebral under-perfusion and oliguria caused by poor renal perfusion. The mortality is very high, but can be reduced by early revascularisation by PCI.

Acute coronary syndrome (ACS) is the most common cause of cardiogenic shock, mainly through a large zone of myocardial ischaemia or a mechanical complication of myocardial infarction. Although uncommon, the short-term mortality of cardiogenic shock is up to 40 % contrasting with a good quality of life in patients discharged alive. An early invasive strategy (i.e. primary PCI, PCI early after fibrinolysis) is indicated for those patients who are suitable for revascularisation. Even if commonly used in clinical practice, there is no evidence supporting the use of IABP in cardiogenic shock.

Suspect right ventricular infarction in patients with inferior infarction, clinical shock and clear lung fields. ST segment elevation ≥ 1 mm in lead V4R is a useful indicator of right ventricular infarction. These patients have an in-hospital mortality of up to 30 % and many benefit greatly from reperfusion therapy. Avoid nitrates and other vasodilators, and treat hypotension with intravenous fluids.

5.6. Other cardiac arrhythmia

The treatment of other cardiac arrhythmia will be covered in more detail in chapter 11.

When atrial fibrillation occurs in the context of an ACS it usually indicates some degree of left ventricular failure: treatment should address that as well as focusing on control of heart rate or rhythm.

When AV-block occurs in the context of acute inferior wall myocardial infarction there is often excess vagal activity. QRS complexes are often narrow and heart rates may not be excessively slow. Treat symptomatic bradycardia in this setting with atropine and if necessary theophylline, and consider temporary cardiac pacing only if bradycardia and hypotension persist after atropine therapy. Complete AV-block in this setting is usually transient and permanent cardiac pacing is often not necessary.

When AV-block occurs in the context of acute anterior myocardial infarction this usually implies extensive myocardial injury and a poor prognosis. The QRS complexes are usually broad and the heart rate is usually slow and resistant to atropine. Temporary cardiac pacing is usually needed and should not be delayed. Many of those who survive this situation will require a permanent pacemaker.

5.7. Cardiac rehabilitation

In all patients after an ACS, an effective programme of cardiac rehabilitation can speed the return to normal activity and encourage measures that will reduce future risk *(see below)*. There is evidence that effective cardiac rehabilitation reduces the need for readmission to hospital. Cardiac rehabilitation is a continuous process, beginning in the cardiac care unit and progressing through to a community-based approach to lifestyle modification and secondary prevention.

5.8. Preventive interventions

Preventive interventions in patients presenting with ACS should be initiated early after hospital admission and should be continued if already in place. Preventive measures improve prognosis by reducing the number of major adverse cardiac events. Prevention with drugs encompasses beta-blockers, angiotensin converting enzyme (ACE) inhibitors/angiotensin receptor blockers (ARB) and statins, as well as basic treatment with ASA and, if indicated, thienopyridines.

5.8.1. Beta-blockers

There is no evidence to support routine intravenous beta-blockers in the pre-hospital or initial ED settings. Early IV use of beta-blockers is contraindicated in patients with clinical signs of hypotension or congestive heart failure. It may be indicated in special situations such as severe hypertension or tachyarrhythmias in the absence of contraindications. It is reasonable to start oral beta-blockers at low doses only after the patient is stabilised.

5.8.2. Other anti-arrhythmics

Apart from beta-blockers, there is no evidence to support the use of anti-arrhythmic prophylaxis after ACS. Ventricular fibrillation (VF) accounts for most of the early deaths from ACS; the incidence of VF is highest in the first hours after onset of symptoms. The effects of antiarrhythmic drugs (lidocaine, magnesium, disopyramide, mexiletine, verapamil, sotalol, tocainamide) given prophylactically to patients with ACS have been studied. Prophylaxis with lidocaine reduces the incidence of VF but may increase mortality. Routine treatment with magnesium in patients with AMI does not reduce mortality. Arrhythmia prophylaxis using disopyramide, mexiletine, verapamil, or other anti-arrhythmics given within the first hours of an ACS does not improve mortality. Therefore prophylactic anti-arrhythmics are not recommended.

5.8.3. Angiotensin-converting enzyme inhibitors, angiotensin receptor blockers

Oral ACE inhibitors reduce mortality when given to patients with AMI with or without early reperfusion therapy. The beneficial effects are most pronounced in patients presenting with anterior infarction, pulmonary congestion or left ventricular ejection fraction < 40 %. Do not give ACE inhibitors if the systolic blood pressure is less than 100 mmHg on admission or if there is a known contraindication to these drugs. Therefore, give an oral ACE inhibitor within 24 h after symptom onset in patients with AMI regardless of whether early reperfusion therapy is planned, particularly in those patients with anterior infarction, pulmonary congestion or a left ventricular ejection fraction below 40 %. Do not give intravenous ACE inhibitors within 24 h of onset of symptoms. Give an angiotensin receptor blocker (ARB) to patients intolerant of ACE inhibitors.

5.8.4. Lipid-lowering therapy

Statins reduce the incidence of major adverse cardiovascular events when given early within the first days after onset an ACS. Consider starting statin therapy in all patients within 24 hours of onset of symptoms of ACS unless contraindicated. If patients are already receiving statin therapy, do not stop it.

Recently published ESC Guidelines stress the importance of beta-blockers as first-line therapy in the management of ventricular arrhythmias (VAs) and the prevention of sudden cardiac death:

> Electrical cardioversion or defibrillation is the intervention of choice to acutely terminate VAs in acute coronary syndrome (ACS) patients. Early (possibly i.v.) administration of beta-blockers can help prevent recurrent arrhythmias. Anti-arrhythmic drug treatment with amiodarone should be considered only if episodes of VT or VF are frequent and can no longer be controlled by successive electrical cardioversion or defibrillation. Intravenous lidocaine may be considered for recurrent

sustained VT or VF not responding to beta-blockers or amiodarone or in the case of contraindications to amiodarone. Recurrent polymorphic VT degenerating into VF may respond to beta-blockers. In addition, deep sedation may be helpful to reduce episodes of VT or VF. The use of other anti-arrhythmic drugs in ACS (e.g. procainamide, propafenone, ajmaline, flecainide) is not recommended.

In patients with LV dysfunction with or without heart failure presenting with sustained VT medical drug therapy for sustained VT should target maximal sympathetic blockade.

KEY LEARNING POINTS

- The acute coronary syndromes comprise unstable angina, non-ST-segment-elevation myocardial infarction, and ST-segment-elevation myocardial infarction.

- Give aspirin, nitroglycerine and morphine to patients presenting with acute coronary syndromes.

- Rapid initial assessment using the history, examination and 12-lead ECG will help to determine the diagnosis and immediate risk.

- Consider immediate reperfusion therapy in those patients with acute myocardial infarction accompanied by ST segment elevation or new LBBB.

- Effective assessment and immediate treatment of patients with acute coronary syndromes will reduce the risk of cardiac arrest and death.

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4

Chapter 5.

In-hospital resuscitation

> **LEARNING OUTCOMES**
>
> **To understand:**
>
> - how to start resuscitation in hospital
> - how to continue resuscitation until more experienced help arrives
> - the importance of high-quality CPR with minimal interruption

1. Introduction

After in-hospital cardiac arrest, the division between basic life support and advanced life support is arbitrary; in practice, the resuscitation process is a continuum. The public expect that clinical staff can undertake cardiopulmonary resuscitation (CPR). For all in-hospital cardiac arrests, ensure that:

- cardiorespiratory arrest is recognised immediately
- help is summoned using a standard telephone number
- CPR is started immediately and, if indicated, defibrillation is attempted as soon as possible (within 3 min at the most).

This chapter is primarily for healthcare professionals who are first to respond to an in-hospital cardiac arrest, but may also be applicable to healthcare professionals working in other clinical settings.

2. Why is in-hospital resuscitation different?

The exact sequence of actions after in-hospital cardiac arrest depends on several factors including:

- location (clinical/non-clinical area; monitored/unmonitored area)
- skills of the first responders
- number of responders
- equipment available
- hospital response system to cardiac arrest and medical emergencies, e.g. medical emergency team (MET), resuscitation team

2.1. Location

In patients who are being monitored closely, cardiorespiratory arrest is usually identified rapidly. Patients in many areas without facilities for close monitoring may have had a period of deterioration and can have an unwitnessed arrest. All patients who are at high risk of cardiac arrest should be cared for in a monitored area where facilities for immediate resuscitation are available. Patients, visitors or staff may also have a cardiac arrest in non-clinical areas (e.g. car parks, corridors). Victims of cardiac arrest may need to be moved to enable effective resuscitation.

2.2. Training of first responders

All healthcare professionals should be able to recognise cardiac arrest, call for help and start resuscitation. Staff should do what they have been trained to do. For example, staff in critical care and emergency medicine may have more advanced resuscitation skills and greater experience in resuscitation than those who are not involved regularly in resuscitation in their normal clinical role. Hospital staff who respond to a cardiac arrest may have different levels of skill to manage the airway, breathing and circulation. Rescuers must use the skills for which they are trained.

2.3. Number of responders

The single responder must always ensure that help is coming. Usually, other staff are nearby and several actions can be undertaken simultaneously. Hospital staffing tends to be at its lowest during the night and at weekends. This may influence patient monitoring, treatment and outcomes. Studies show that survival rates from in-hospital cardiac arrest are lower during nights and weekends.

2.4. Equipment available

Staff in all clinical areas should have immediate access to resuscitation equipment and drugs to facilitate rapid resuscitation of the patient in cardiorespiratory arrest. Ideally, the equipment used for cardiopulmonary resuscitation (including defibrillators) and the layout of equipment and drugs should be standardised throughout the hospital. You should be familiar with the resuscitation equipment used in your clinical area.

A review by the Resuscitation Council (UK) of serious patient safety incidents associated with CPR and patient deterioration reported to the National Patient Safety Agency showed that equipment problems during resuscitation (e.g. equipment missing or not working) is common. All resuscitation equipment needs to be checked on a regular basis to ensure it is ready for use. AEDs should be considered for clinical and non-clinical areas where staff do not have rhythm recognition skills or rarely need to use a defibrillator.

After successful resuscitation, patients may need transferring to other clinical areas (e.g. intensive care unit) or other hospitals. Transfer equipment and drugs should be available to enable this. This should include waveform capnography for those patients have had tracheal intubation and are ventilated *(see chapter 7)*.

Figure 5.1
In-hospital resuscitation algorithm

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                    ┌─────────────────────────────┐
                    │   Collapsed/sick patient    │
                    └─────────────────────────────┘
                                  │
                    ┌─────────────────────────────┐
                    │ Shout for HELP & assess     │
                    │         patient             │
                    └─────────────────────────────┘
                                  │
        ┌──────┐    ┌─────────────────────────────┐    ┌──────┐
        │  No  │◄──►│       Signs of life?        │◄──►│ Yes  │
        └──────┘    └─────────────────────────────┘    └──────┘
           │                                               │
  ┌──────────────────┐                         ┌──────────────────┐
  │ Call             │                         │ Assess ABCDE     │
  │ resuscitation    │                         │ Recognise & treat│
  │ team             │                         │ Oxygen,          │
  └──────────────────┘                         │ monitoring,      │
           │                                   │ IV access        │
  ┌──────────────────┐                         └──────────────────┘
  │ CPR 30:2         │                                   │
  │ with oxygen and  │                         ┌──────────────────┐
  │ airway adjuncts  │                         │ Call             │
  └──────────────────┘                         │ resuscitation    │
           │                                   │ team if          │
  ┌──────────────────┐                         │ appropriate      │
  │ Apply pads/      │                         └──────────────────┘
  │ monitor          │                                   │
  │ Attempt          │                         ┌──────────────────┐
  │ defibrillation   │                         │ Handover to      │
  │ if appropriate   │                         │ resuscitation    │
  └──────────────────┘                         │ team             │
           │                                   └──────────────────┘
  ┌──────────────────┐
  │ Advanced Life    │
  │ Support when     │
  │ resuscitation    │
  │ team arrives     │
  └──────────────────┘
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2.5. Resuscitation team

The resuscitation team may take the form of a traditional cardiac arrest team, which is called only when cardiac arrest is recognised. Alternatively, hospitals may have strategies to recognise patients at risk of cardiac arrest and summon a team (e.g. MET) before cardiac arrest occurs. The term resuscitation team reflects the range of response teams. In-hospital cardiac arrests are rarely sudden or unexpected. A strategy of recognising patients at risk of cardiac arrest may enable some of these arrests to be prevented or prevent futile resuscitation attempts in those who are unlikely to benefit from CPR *(chapter 3)*.

Resuscitation teams rarely have formal pre- and post event briefings (briefings and debriefings) to plan roles and actions during resuscitations. Resuscitation team members should meet for introductions and plan before they attend actual events. Team members should also debrief after each event based on what they actually did during the resuscitation. This should ideally be based on data collected during the event.

3. Sequence for collapsed patient in a hospital

An algorithm for the initial management of in-hospital cardiac arrest is shown in figure 5.1.

3.1. Ensure personal safety

There are very few reports of harm to rescuers during resuscitation.

- Your personal safety and that of resuscitation team members is the first priority during any resuscitation attempt.

- Check that the patient's surroundings are safe.

- Put on gloves as soon as possible. Other protective measures, such as eye protection, aprons and face masks, may be necessary.

- The risk of infection is much lower than perceived. There are isolated reports of infections such as tuberculosis (TB), and severe acute respiratory distress syndrome (SARS). Transmission of HIV during CPR has never been reported.

- Wear full personal protective equipment (PPE) when the victim has a serious infection such as TB or SARS. Follow local infection control measures to minimise risks.

- Be careful with sharps; a sharps box must be available. Use safe handling techniques for moving victims during resuscitation.

- Take care with patients exposed to poisons. Avoid mouth-to-mouth ventilation and exhaled air in hydrogen cyanide or hydrogen sulphide poisoning.

- Avoid contact with corrosive chemicals (e.g. strong acids, alkalis, paraquat) or substances such as organophosphates that are easily absorbed through the skin or respiratory tract.

- There are no reports of infection acquired during CPR training. Nevertheless, take sensible precautions to minimise potential cross-infection from manikins. Clean manikins regularly and disinfect thoroughly after each use. Some manikins have disposable face pieces and airways to simplify cleaning.

3.2. Check the patient for a response

- If you see a patient collapse or find a patient apparently unconscious first shout for help, then assess if he is responsive (shake and shout). Gently shake his shoulders and ask loudly: "Are you all right?" *(figure 5.2)*.

- If other members of staff are nearby it will be possible to undertake actions simultaneously.

Figure 5.2
Shake and shout

3.3 A. If he responds

- Urgent medical assessment is required. Call for help according to local protocols. This may be a resuscitation team (e.g. MET).

- While waiting for the team, assess the patient using the ABCDE (Airway, Breathing, Circulation, Disability, Exposure) approach.

- Give the patient oxygen - use pulse oximetry to guide oxygen therapy.

- Attach monitoring (minimum pulse oximetry, ECG and blood pressure) and record vital signs.

- Obtain venous access.

- Prepare for handover to team using SBAR (Situation, Background, Assessment, Recommendation) or RSVP (Reason, Story, Vital signs, Plan).

Figure 5.3
Head tilt and chin lift

Figure 5.4
Looking for breathing and any other movement

3.3 B. If he does not respond

- The exact sequence will depend on your training and experience in assessment of breathing and circulation in sick patients. Agonal breathing (occasional gasps, slow, laboured or noisy breathing) is common in the early stages of cardiac arrest and is a sign of cardiac arrest and should not be confused as a sign of life.

- Shout for help (if not already).

- Turn the patient on to his back.

- Open the airway using head tilt and chin lift *(figure 5.3)*.

- If there is a risk of cervical spine injury, establish a clear upper airway by using jaw-thrust or chin lift in combination with manual in-line stabilisation (MILS) of the head and neck by an assistant (if enough personnel are available). If life-threatening airway obstruction persists despite effective application of jaw-thrust or chin lift, add head tilt a small amount at a time until the airway is open; establishing a patent airway, oxygenation and ventilation takes priority over concerns about a potential cervical spine injury.

Keeping the airway open, look, listen, and feel *(figure 5.4)* to determine if the victim is breathing normally. This is a rapid check and should take **less than 10 seconds** (an occasional gasp, slow, laboured or noisy breathing is not normal):

- Look for chest movement

- Listen at the victim's mouth for breath sounds

- Feel for air on your cheek

- Check for signs of a circulation:

  - It may be difficult to be certain that there is no pulse. If the patient has no signs of life (consciousness, purposeful movement, normal breathing, or coughing), or if there is doubt, start CPR immediately until more experienced help arrives or the patient shows signs of life.

  - Delivering chest compressions to a patient with a beating heart is unlikely to cause harm. However, delays in diagnosing cardiac arrest and starting CPR will adversely effect survival and must be avoided.

  - Only those experienced in ALS should try to assess the carotid pulse whilst simultaneously looking for signs of life *(figure 5.5)*. This rapid assessment should take no more than 10 seconds. Start CPR if there is any doubt about the presence or absence of a pulse.

- If the patient has no signs of life, no pulse, or if there is any doubt, start CPR immediately.

- Assess the patient to confirm cardiac arrest even if the patient is monitored in a critical care area.

5

Figure 5.5
Simultaneous check for breathing and carotid pulse

3.4 A. If the patient shows signs of life or a pulse is palpable

- Urgent medical assessment is required. Depending on the local protocols, this may take the form of a resuscitation team. While awaiting this team, assess the patient using the ABCDE approach, give oxygen, attach monitoring, and insert an intravenous cannula.

- Follow the steps in 3A above whilst waiting for the team.

- The patient is at high risk of further deterioration and cardiac arrest and needs continued observation until the team arrives.

3.4 B. If the patient doesn't show signs of life or and no pulse is palpable

- One person starts CPR as others call the resuscitation team and collect the resuscitation equipment and a defibrillator. If only one member of staff is present, this will mean leaving the patient.

- Give 30 chest compressions followed by 2 ventilations.

- Compress to a depth of approximately 5 cm but not more than 6 cm.

- Chest compressions should be performed at a rate of 100–120 min^{-1}.

- Allow the chest to recoil completely after each compression; do not lean on the chest.

- Minimise interruptions and ensure high-quality compressions.

- Undertaking high-quality chest compressions for a prolonged time is tiring; with minimal interruption, try to change the person doing chest compressions every 2 minutes.

- Maintain the airway and ventilate the lungs with the most appropriate equipment immediately to hand. Pocket mask ventilation or two-rescuer bag-mask ventilation, which can be supplemented with an oral airway, should be started. Alternatively, use a supraglottic airway device (SGA) and self-inflating bag. Tracheal intubation should be attempted only by those who are trained, competent and experienced in this skill.

- Waveform capnography must be used for confirming tracheal tube placement and monitoring ventilation rate. Waveform capnography can also be used with a bag-mask device and SGA. The further use of waveform capnography to monitor CPR quality and potentially identify ROSC during CPR is discussed later.

- Use an inspiratory time of 1 second and give enough volume to produce a normal chest rise. Add supplemental oxygen to give the highest feasible inspired oxygen as soon as possible.[4]

- Once the patient's trachea has been intubated or a SGA has been inserted, continue uninterrupted chest compressions (except for defibrillation or pulse checks when indicated) at a rate of 100 to 120 min^{-1} and ventilate the lungs at approximately 10 breaths min^{-1}. Avoid hyperventilation (both excessive rate and tidal volume).

- If there is no airway and ventilation equipment available, consider giving mouth-to-mouth ventilation. If there are clinical reasons to avoid mouth-to-mouth contact, or you are unable to do this, do chest compressions until help or airway equipment arrives.

- When the defibrillator arrives, apply self-adhesive defibrillation pads to the patient whilst chest compressions continue and then briefly analyse the rhythm. If self-adhesive defibrillation pads are not available, use paddles. The use of self-adhesive electrode pads or a 'quick-look' paddles technique will enable rapid assessment of the heart rhythm compared with attaching ECG electrodes. Pause briefly to assess the heart rhythm. With a manual defibrillator, if the rhythm is VF/pVT charge the defibrillator while another rescuer continues chest compressions. Once the defibrillator is charged, pause the chest compressions and then give one shock, and immediately resume chest compressions. Ensure no one is touching the patient during shock delivery. Plan and ensure safe defibrillation before the planned pause in chest compressions.

- If using an automated external defibrillator (AED) follow the AED's audio-visual prompts, and similarly aim to minimise pauses in chest compressions by rapidly following prompts.

- In settings where self-adhesive defibrillation pads are not available, alternative defibrillation strategies using paddles are used to minimise the preshock pause.

- Restart chest compressions immediately after the defibrillation attempt. Minimise interruptions in chest compressions. When using a manual defibrillator it is possible to reduce the pause between stopping and restarting of chest compressions to less than five seconds.

- Continue resuscitation until the resuscitation team arrives or the patient shows signs of life. Follow the voice prompts if using an AED.

- Once resuscitation is underway, and if there are sufficient staff present, prepare intravenous cannulae and drugs likely to be used by the resuscitation team (e.g. adrenaline).

- Identify one person to be responsible for handover to the resuscitation team leader. Use a structured communication tool for handover (e.g. SBAR, RSVP). Locate the patient's records.

- The quality of chest compressions during in-hospital CPR is frequently sub-optimal. The importance of uninterrupted chest compressions cannot be overemphasised. Even short interruptions in chest compressions are disastrous for outcome and every effort must be made to ensure that continuous, effective chest compression is maintained throughout the resuscitation attempt. Chest compressions should commence at the beginning of a resuscitation attempt and continue uninterrupted unless they are paused briefly for a specific intervention (e.g. rhythm check). Most interventions can be performed without interruptions in chest compressions. The team leader should monitor the quality of CPR and alternate CPR providers if the quality of CPR is poor.

- Continuous $ETCO_2$ monitoring during CPR can be used to indicate the quality of CPR, and a significant rise in $ETCO_2$ can be an indicator of ROSC during chest compressions.

- If possible, the person providing chest compressions should be changed every 2 minutes, but without pauses in chest compressions.

Figure 5.6
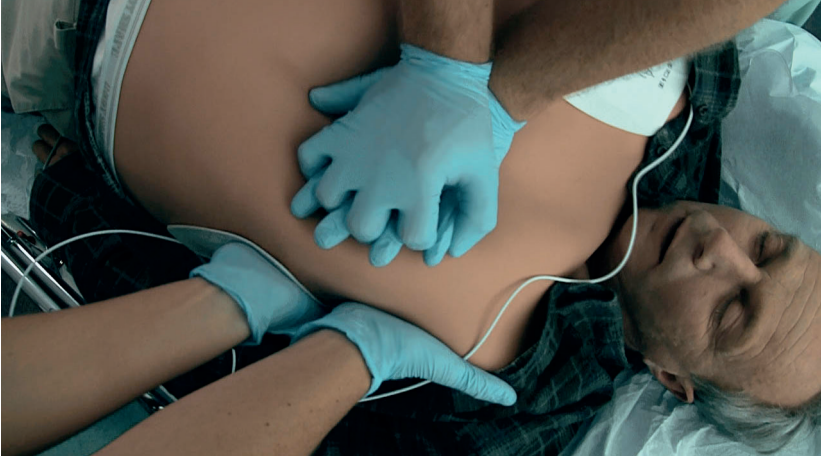Call the resuscitation team

Figure 5.7
Hand position for chest compressions

Figure 5.8
Hands placed in the middle of the lower half of the sternum

5

Figure 5.9
Maintain chest compressions while self-adhesive pads are applied

3.4 C. If he is not breathing and has a pulse (respiratory arrest)

- Ventilate the patient's lungs (as described above) and check for a pulse every 10 breaths (about every minute).

- This diagnosis can be made only if you are confident in assessing breathing and pulse or the patient has other signs of life (e.g. warm and well perfused, normal capillary refill).

- If there are any doubts about the presence of a pulse, start chest compressions until more experienced help arrives.

- All patients in respiratory arrest will develop cardiac arrest if the respiratory arrest is not treated rapidly and effectively.

4. Audit of cardiac arrests

All in-hospital cardiac arrests should be reviewed and audited using a national data collection system. These databases monitor and report on the incidence of and outcome from, cardiac arrests in order to inform practice and policy. They aim to identify and foster improvements in the prevention, care delivery and outcomes from cardiac arrest. Participating in these audits means that your organisation is collecting and contributing to national, standardised data on cardiac arrest, enabling improvements in patient care.

> **KEY LEARNING POINTS**
>
> - The exact sequence of actions after in-hospital cardiac arrest depends on the location, skills of the first responders, number of responders, equipment available, and the hospital response system to cardiac arrest and medical emergencies.
>
> - Deliver high-quality chest compressions. Compress to a depth of approximately 5 cm but not more than 6 cm, rate of 100-120 min^{-1}, and allow complete recoil between compressions.
>
> - Minimise interruptions in chest compressions for other interventions – this means all interruptions must be planned before stopping compressions.

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Chapter 6.

Advanced Life Support algorithm

LEARNING OUTCOMES

To understand:

- the function of the advanced life support (ALS) algorithm
- the importance of minimally interrupted high-quality chest compressions
- the treatment of shockable and non-shockable rhythms
- when and how to give drugs during cardiac arrest
- the potentially reversible causes of cardiac arrest

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1.   Introduction

Heart rhythms associated with cardiac arrest are divided into two groups: shockable rhythms (ventricular fibrillation/pulseless ventricular tachycardia (VF/pVT)) and non-shockable rhythms (asystole and pulseless electrical activity (PEA)). The principle difference in the management of these two groups of arrhythmias is the need for attempted defibrillation in patients with VF/pVT. Subsequent actions, including chest compressions, airway management and ventilation, venous access, injection of adrenaline and the identification and correction of reversible factors, are common to both groups.

The ALS algorithm *(figure 6.1)* is a standardised approach to cardiac arrest management. This has the advantage of enabling treatment to be delivered expediently, without protracted discussion. It enables each member of the resuscitation team to predict and prepare for the next stage in the patient's treatment, further enhancing efficiency of the team. Although the ALS algorithm is applicable to most cardiac arrests, additional interventions may be indicated for cardiac arrest caused by special circumstances *(see chapter 12)*.

The interventions that unquestionably contribute to improved survival after cardiac arrest are prompt and effective bystander cardiopulmonary resuscitation (CPR), uninterrupted, high-quality chest compressions, and early defibrillation for VF/pVT. The use of adrenaline

has been shown to increase return of spontaneous circulation (ROSC), but no resuscitation drugs or advanced airway interventions have been shown to increase survival to hospital discharge after cardiac arrest. Thus, although drugs and advanced airways are still included among ALS interventions, they are of secondary importance to high-quality, uninterrupted chest compressions and early defibrillation.

2. Shockable rhythms (VF/pVT)

The first monitored rhythm is VF/pVT in approximately 20 % of cardiac arrests, both in- and out-of-hospital. VF/pVT will also occur at some stage during resuscitation in about 25 % of cardiac arrests with an initial documented rhythm of asystole or PEA.

2.1. Treatment of shockable rhythms (VF/pVT)

1. Confirm cardiac arrest - check for signs of life or if trained to do so, breathing and pulse simultaneously.

2. Call resuscitation team.

3. Perform uninterrupted chest compressions while applying self-adhesive defibrillation/monitoring pads - one below the right clavicle and the other in the V6 position in the midaxillary line.

4. Plan actions before pausing CPR for rhythm analysis and communicate these to the team.

5. Stop chest compressions not longer than 2 seconds to check rhythm, Resume chest compressions immediately.

6. Confirm VF/pVT, if in doubt use a print out rhythm strip; the designated person selects the appropriate energy on the defibrillator (150-200 J biphasic for the first shock and 150-360 J biphasic for subsequent shocks) and presses the charge button.

7. While the defibrillator is charging, warn all rescuers other than the individual performing the chest compressions to "stand clear" and remove any oxygen delivery device as appropriate. Ensure that the rescuer giving the compressions is the only person touching the patient.

8. Once the defibrillator is charged, tell the rescuer doing the chest compressions to "stand clear"; when clear, give the shock.

9. Without reassessing the rhythm or feeling for a pulse, restart CPR using a ratio of 30:2, starting with chest compressions.

10. Continue CPR for 2 min; the team leader prepares the team for the next pause in CPR.

11. Pause briefly to check the monitor.

12. If VF/pVT, repeat steps 6-11 above and deliver a second shock.

13. If VF/pVT persists repeat steps 6-8 above and deliver a third shock. Without reassessing the rhythm or feeling for a pulse, resume CPR (30:2) immediately after the shock, starting with chest compressions.

14. If IV/IO access has been obtained, during the next 2 minutes of CPR give adrenaline 1 mg and amiodarone 300 mg.

15. The use of waveform capnography may enable ROSC to be detected without pausing chest compressions and may be used as a way of avoiding a bolus injection of adrenaline after ROSC has been achieved. If ROSC is suspected during CPR withhold adrenaline. Give adrenaline if cardiac arrest is confirmed at the next rhythm check.

16. Give further adrenaline 1 mg IV after alternate shocks (i.e., in practice, this will be about once every two cycles of the algorithm).

Figure 6.1
Shock delivery

6
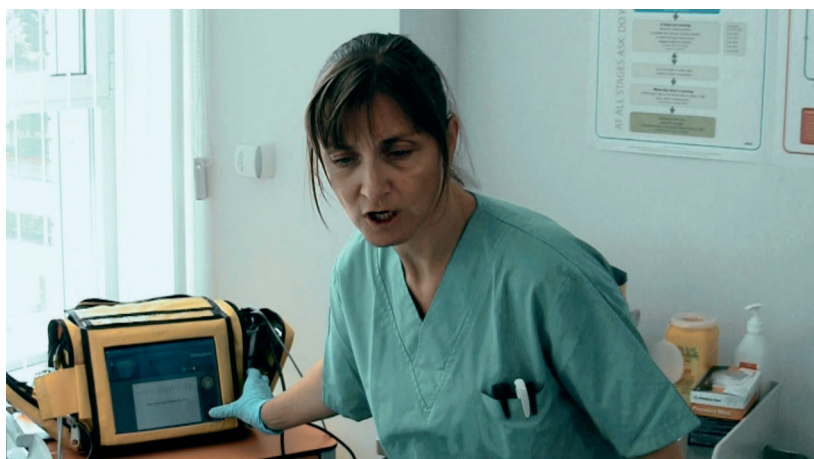
If signs of life return during CPR (purposeful movement, normal breathing or coughing), or there is a significant increase in ETCO$_2$, check the monitor.

If organised electrical activity compatible with a cardiac output is seen during a rhythm check, seek evidence of ROSC:

- Check a central pulse and end-tidal (ETCO$_2$) trace if available.

- If there is evidence of ROSC, start post-resuscitation care.

- If no signs of ROSC, continue CPR and switch to the non-shockable algorithm.

If asystole is confirmed during a rhythm check continue CPR and switch to the non-shockable algorithm.

Figure 6.2
Adult advanced life support algorithm

Unresponsive and not breathing normally?

Call Resuscitation Team

CPR 30:2
Attach defibrillator/monitor
Minimise interruptions

Assess rhythm

Shockable (VF/Pulseless VT)

Non-shockable (PEA/Asystole)

1 Shock
Minimise interruptions

Return of spontaneous circulation

Immediately resume CPR for 2 min
Minimise interruptions

Immediately resume CPR for 2 min
Minimise interruptions

IMMEDIATE POST CARDIAC ARREST TREATMENT
- Use ABCDE approach
- Aim for SaO_2 of 94-98 %
- Aim for normal $PaCO_2$
- 12 Lead ECG
- Treat precipitating cause
- Targeted temperature management

DURING CPR
- Ensure high-quality chest compressions
- Minimise interruptions to compressions
- Give oxygen
- Use waveform capnography
- Continuous compressions when advanced airway in place
- Vascular access (intravenous or intraosseous)
- Give adrenaline every 3-5 min
- Give amiodarone after 3 shocks

TREAT REVERSIBLE CAUSES
Hypoxia
Hypovolaemia
Hypo-/hyperkalaemia/metabolic
Hypothermia/hyperthermia
Thrombosis – coronary or pulmonary
Tension pneumothorax
Tamponade – cardiac
Toxins

CONSIDER
- Ultrasound imaging
- Mechanical chest compressions to facilitate transfer/treatment
- Coronary angiography and percutaneous coronary intervention
- Extracorporeal CPR

The interval between stopping compressions and delivering a shock must be minimised and, ideally, should not exceed 5 s. Longer interruptions in chest compressions reduce the chance of a shock restoring spontaneous circulation.

Chest compressions are resumed immediately after a shock without checking the rhythm or a pulse because even if the defibrillation attempt is successful in restoring a perfusing rhythm. It is very rare for a pulse to be palpable immediately after defibrillation and the delay in trying to palpate a pulse will further compromise the myocardium if a perfusing rhythm has not been restored. If a perfusing rhythm has been restored, giving chest compressions does not increase the chance of VF recurring. In the presence of post-shock asystole chest compressions may usefully induce VF.

If ROSC has not been achieved with this 3rd shock, the adrenaline may improve myocardial blood flow and increase the chance of successful defibrillation with the next shock. Despite the widespread use of adrenaline during resuscitation, there is no placebo-controlled study that shows that the routine use of any vasopressor at any stage during human cardiac arrest increases neurologically intact survival to hospital discharge. Current evidence is insufficient to support or refute the routine use of any particular drug or sequence of drugs. Despite the lack of human data, the use of adrenaline is still recommended, based largely on animal data and increased short-term survival in humans.

Timing of adrenaline dosing can cause confusion amongst ALS providers and this aspect needs to be emphasised during training. Training should emphasise that giving drugs must not lead to interruptions in CPR and delay interventions such as defibrillation. The first dose of adrenaline is given during the 2 minutes period following delivery of the third shock; amiodarone 300 mg may also be given after the third shock. Do not stop CPR to check the rhythm before giving drugs unless there are clear signs of ROSC.

Figure 6.3
Shock delivery
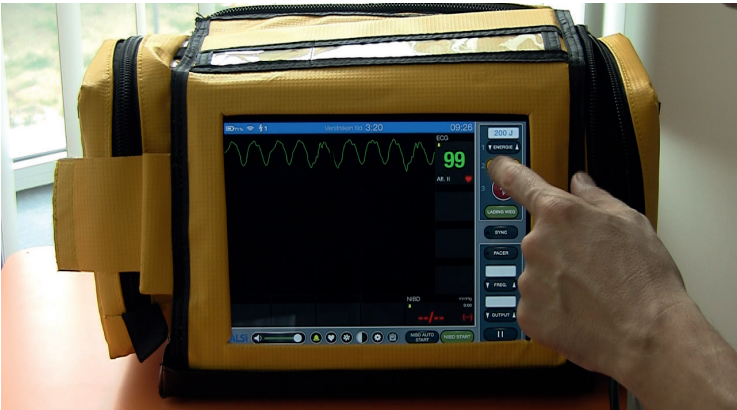
Subsequent doses of adrenaline are given after alternate 2-minute cycles of CPR (which equates to every 3-5 min) for as long as cardiac arrest persists. If VF/pVT persists, or recurs, give a further dose of 150 mg amiodarone. Lidocaine, 1 mg kg^{-1}, may be used as an alternative if amiodarone is not available, but do not give lidocaine if amiodarone has been given already.

When the rhythm is checked 2 min after giving a shock, if a non-shockable rhythm is present and the rhythm is organised (complexes appear regular or narrow), try to palpate a central pulse and look for other evidence of ROSC (e.g. sudden increase in ETCO$_2$ or evidence of cardiac output on any invasive monitoring equipment). Rhythm checks must be brief, and pulse checks undertaken only if an organised rhythm is observed. If an organised rhythm is seen during a 2-minute period of CPR, do not interrupt chest compressions to palpate a pulse unless the patient shows signs of life suggesting ROSC. If there is any doubt about the presence of a pulse in the presence of an organised rhythm, resume CPR. If the patient has ROSC, begin post-resuscitation care. If the patient's rhythm changes to asystole or PEA, see non-shockable rhythms below.

It is important in shock-refractory VF/pVT to check the position and contact of the defibrillation pads. The duration of any individual resuscitation attempt is a matter of clinical judgement, and should take into account the perceived prospect of a successful outcome. If it was considered appropriate to start resuscitation, it is usually considered worthwhile continuing as long as the patient remains in identifiable VF/pVT.

If there is doubt about whether the rhythm is asystole or very fine VF, do not attempt defibrillation; instead, continue chest compressions and ventilation. Very fine VF that is difficult to distinguish from asystole is unlikely to be shocked successfully into a perfusing rhythm. Continuing high-quality CPR may improve the amplitude and frequency of the VF and improve the chance of subsequent successful defibrillation to a perfusing rhythm. Delivering repeated shocks in an attempt to defibrillate what is thought to be very fine VF will increase myocardial injury both directly from the electric current and indirectly from the interruptions in coronary blood flow. If the rhythm is clearly VF, attempt defibrillation.

2.1.1. Precordial thump

A single precordial thump has a very low success rate for cardioversion of a shockable rhythm. Its routine use is therefore not recommended. It may be appropriate therapy only when used without delay whilst awaiting the arrival of a defibrillator in a monitored VF/pVT arrest. Using the ulnar edge of a tightly clenched fist, deliver a sharp impact to the lower half of the sternum from a height of about 20 cm, then retract the fist immediately to create an impulse-like stimulus. There are rare reports of a precordial thump converting a perfusing to a non-perfusing rhythm.

2.1.2. Witnessed, monitored VF/pVT in specific settings

If a patient has a monitored and **witnessed** cardiac arrest in the catheter laboratory, coronary care unit, a critical care area or whilst **monitored** after cardiac surgery, and a manual defibrillator is rapidly available:

- Confirm cardiac arrest and shout for help.

- If the initial rhythm is VF/pVT, give up to three quick successive (stacked) shocks.

- Rapidly check for a rhythm change and, if appropriate, ROSC after each defibrillation attempt.

- Start chest compressions and continue CPR for two minutes if the third shock is unsuccessful. With respect to the ALS algorithm, these three quick, successive shocks are regarded as the first shock.

This three-shock strategy may also be considered for an initial, witnessed VF/pVT cardiac arrest if the patient is already connected to a manual defibrillator - these circumstances are rare. There are no data supporting a three-shock strategy in any of these circumstances, but it is unlikely that chest compressions will improve the already very high chance of ROSC when defibrillation occurs early in the electrical phase, immediately after onset of VF.

2.2. Non-shockable rhythms (PEA and asystole)

Pulseless electrical activity (PEA) is defined as organised cardiac electrical activity in the absence of any palpable pulses. These patients often have some mechanical myocardial contractions but they are too weak to produce a detectable pulse or blood pressure. PEA may be caused by reversible conditions that can be treated *(see below)*. Survival following cardiac arrest with asystole or PEA is unlikely unless a reversible cause can be found and treated quickly and effectively.

Asystole is the absence of electrical activity on the ECG trace. During CPR, ensure the ECG pads are attached to the chest and the correct monitoring mode is selected. Ensure the gain setting is appropriate. Whenever a diagnosis of asystole is made, check the ECG carefully for the presence of P waves because in this situation ventricular standstill may be treated effectively by cardiac pacing. Attempts to pace true asystole are unlikely to be successful.

Treatment for PEA and asystole

1. Start CPR 30:2.

2. If asystole is displayed, without stopping CPR, check that the leads are attached correctly.

3. Once an advanced airway has been sited, continue chest compressions without pausing during ventilation.

4. Give adrenaline 1 mg as soon as venous or intraosseous access is achieved, and repeat every alternate CPR cycle (i.e. about every 3-5 minutes).

5. After 2 minutes of CPR, recheck the rhythm. If asystole is present, resume CPR immediately.

6. If an organised rhythm is present, attempt to palpate a pulse.

7. If a pulse and/or signs of life are present, start post resuscitation care.

8. If no pulse and/or no signs of life are present (PEA):

   - Continue CPR.

   - Recheck the rhythm after 2 min and proceed accordingly.

   - Give further adrenaline 1 mg IV every 3-5 min (during alternate 2-min cycles of CPR).

   - If VF/pVT at rhythm check, change to shockable side of algorithm.

9. If asystole or an agonal rhythm is seen at rhythm check:

   - Continue CPR.

   - Recheck the rhythm after 2 min and proceed accordingly.

   - Give further adrenaline 1 mg IV every 3-5 min (during alternate 2-min cycles of CPR).

Whenever a diagnosis of asystole is made, check the ECG carefully for the presence of P waves, because this may respond to cardiac pacing. There is no benefit in attempting to pace true asystole.

3.   During CPR

During the treatment of persistent VF/pVT or PEA/asystole, emphasis is placed on high-quality chest compressions between defibrillation attempts, recognising and treating reversible causes (4 Hs and 4 Ts), obtaining a secure airway, and vascular access.

During CPR with a 30:2 ratio, the underlying rhythm may be seen clearly on the monitor as compressions are paused to enable ventilation. If VF is seen during this brief pause (whether on the shockable or non-shockable side of the algorithm), do not attempt defibrillation at this stage; instead, continue with CPR until the 2-minute period is completed. Knowing that the rhythm is VF, the team should be fully prepared to deliver a shock with minimal delay at the end of the 2-minute period of CPR.

3.1.   Maintain high-quality, uninterrupted chest compressions

The quality of chest compressions and ventilations are important determinants of outcome, yet are frequently performed poorly by healthcare professionals. Avoid interruptions in chest compressions because pauses cause coronary perfusion pressure to decrease substantially. Ensure compressions are of adequate depth (approximately 5 cm but not more than 6 cm) and rate (100-120 min^{-1}), and release pressure from the chest completely between compressions.

As soon as the airway is secured, continue chest compressions without pausing during ventilation. To reduce fatigue, change the individual undertaking compressions every 2 min or earlier if necessary. Use CPR feedback/prompt devices when available. Be aware that some devices may fail to compensate for compression of the underlying mattress during CPR on a bed when providing feedback.

3.2. Airway and ventilation

Tracheal intubation provides the most reliable airway, but should be attempted only if the healthcare provider is properly trained and has regular, ongoing experience with the technique. Tracheal intubation must not delay defibrillation attempts. Personnel skilled in advanced airway management should attempt laryngoscopy and intubation without stopping chest compressions; a brief pause in chest compressions may be required as the tube is passed through the vocal cords, but this pause should be less than 5 seconds. Alternatively, to avoid any interruptions in chest compressions, the intubation attempt may be deferred until ROSC.

No studies have shown that tracheal intubation increases survival after cardiac arrest. After intubation, confirm correct tube position, ideally with waveform capnography and secure it adequately. Ventilate the lungs at 10 breaths min^{-1}; do not hyperventilate the patient. Once the patient's trachea has been intubated, continue chest compressions, at a rate of 100-120 min^{-1} without pausing during ventilation. A pause in the chest compressions causes the coronary perfusion pressure to fall substantially. On resuming compressions, there is some delay before the original coronary perfusion pressure is restored, thus chest compressions that are not interrupted for ventilation (or any reason) result in a substantially higher mean coronary perfusion pressure.

In the absence of personnel skilled in tracheal intubation, a supraglottic airway (SGA) (e.g. laryngeal mask airway, laryngeal tube or i-gel) is an acceptable alternative. Once a SGA has been inserted, attempt to deliver continuous chest compressions, uninterrupted by ventilation. If excessive gas leakage causes inadequate ventilation of the patient's lungs, chest compressions will have to be interrupted to enable ventilation (using a ratio of 30:2).

3.3. Vascular access

Obtain intravenous access if this has not been done already. Although peak drug concentrations are higher and circulation times are shorter when drugs are injected into a central venous catheter compared with a peripheral cannula, insertion of a central venous catheter requires interruption of CPR and is associated with several potential complications. Peripheral venous cannulation is quicker, easier, and safer. Drugs injected peripherally must be followed by a flush of at least 20 ml of fluid and elevation of the extremity for 10-20 s to facilitate drug delivery to the central circulation. If intravenous access cannot be established within the first 2 min of resuscitation, consider gaining intraosseous (IO) access *(figure 6.4)*. Tibial and humeral sites are readily accessible and provide equal flows for fluids. Intraosseous delivery of resuscitation drugs will achieve adequate plasma concentrations. Several studies indicate that IO access is safe and effective for fluid resuscitation and drug delivery.

6

4. Use of Intraosseous (IO) access during cardiac arrest

4.1. Introduction

Intraosseous (IO) infusion as a means of vascular access has been recognised for close to a century and has seen a resurgence in the last decade particularly for use in resuscitation in adults *(figure 1)*. This is due in part to the publication of a number of studies that suggest it is a viable alternative to intravenous (IV) access but also the development of powered devices for inserting the needle, a technique recently supported by the findings of a systematic review. Intraosseous access is also quicker than central venous access in patients in whom peripheral venous access is not possible. Furthermore, the use of central venous catheters (CVC) during resuscitation requires considerable skill and can lead to prolonged interruptions to chest compressions. Current recommendations are to establish IO access if IV access is not possible or associated with a delay in the first 2 minutes of resuscitation.

4.1.1. Practical considerations

There are 3 main insertion sites recommended for use in adults:

- proximal tibia
- distal tibia
- proximal humerus

The patient should be assessed for the presence of contraindications for the use of IO access. These are:

- fracture or prosthesis in the targeted bone
- recent IO (past 24-48 hrs) in the same limb including previous failed attempt
- signs of infection at insertion site
- inability to locate landmarks

4.1.2. Insertion

Training in the specific device to be used in clinical practice is essential. Site of insertion, identification of landmarks and technique for insertion will differ depending on the device being used. Errors in identification of landmarks or in insertion technique increase the risk of failure and complications.

1. Once inserted, correct placement must be confirmed before delivery of drugs or infusion of fluids. The needle should be aspirated; presence of IO blood indicates correct placement, absence of aspirate does not necessarily imply a failed attempt. There are reports of IO blood being used for laboratory analysis including glucose, haemogolobin and electrolytes. Samples must be labeled as bone marrow aspirate before being sent to the laboratory.

2. The needle should be flushed to ensure patency and observed for leakage or extravasation. This is best achieved using an extension set flushed with 0.9 % saline attached to the hub of the needle before use.

3. Once IO access has been confirmed resuscitation drugs including adrenaline and amiodarone can be infused. Fluids and blood products can also be delivered but pressure will be needed to achieve reasonable flow rates using either a pressure bag or a 50 ml syringe.

4. Manufacturer's guidance should be followed both for securing the needle and the maximum length of time it can be left in place.

Complications associated with IO access/use are:

- extravasations into the soft tissues surrounding the insertion site
- dislodgement of the needle
- embolism
- compartment syndrome due to extravasation
- fracture or chipping of the bone during insertion
- pain related to the infusion of drugs/fluids
- infection/osteomyelitis

6

Figures 6.4
Examples of intraosseous devices
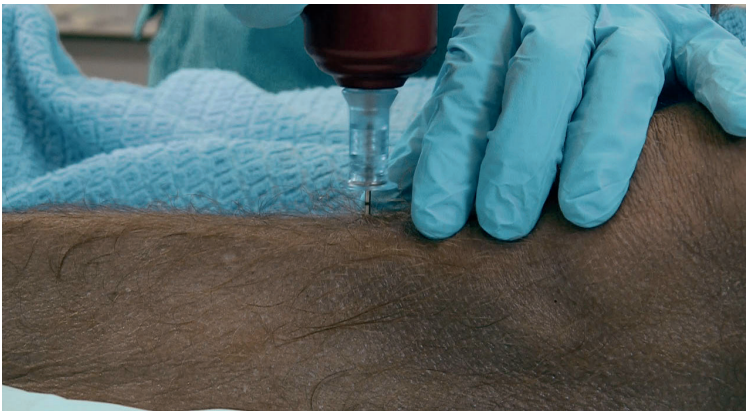
5. Reversible causes

Potential causes or aggravating factors for which specific treatment exists must be considered during any cardiac arrest. For ease of memory, these are divided into two groups of four based upon their initial letter - either H or T *(figure 6.5)*. More details on many of these conditions are covered in chapter 12.

- Hypoxia

- Hypovolaemia

- Hyperkalaemia, hypokalaemia, hypoglycaemia, hypocalcaemia, acidaemia and other metabolic disorders

- Hypothermia

- Tension pneumothorax

- Tamponade

- Toxins

- Thrombosis (pulmonary embolism or coronary thrombosis)

Figure 6.5
The four Hs and four Ts
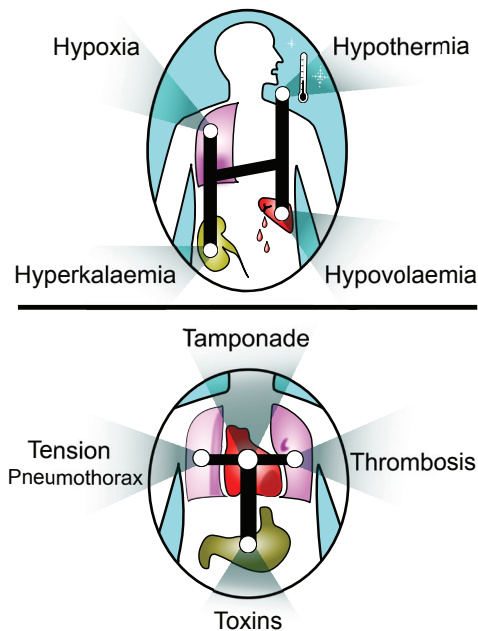
- **The four Hs**

Minimise the risk of **hypoxia** by ensuring that the patient's lungs are ventilated adequately with 100% oxygen. Make sure there is adequate chest rise and bilateral breath sounds. Using the techniques described in chapter 7, check carefully that the tracheal tube is not misplaced in a bronchus or the oesophagus.

Pulseless electrical activity caused by **hypovolaemia** is due usually to severe haemorrhage. Evidence of haemorrhage may be obvious, e.g. trauma *(chapter 12)*, or occult e.g. gastrointestinal bleeding, or rupture of an aortic aneurysm. Intravascular volume should be restored rapidly with fluid and blood, coupled with urgent surgery to stop the haemorrhage.

Hyperkalaemia, hypokalaemia, hypoglycaemia, hypocalcaemia, acidaemia and other metabolic disorders are detected by biochemical tests or suggested by the patient's medical history e.g. renal failure *(chapter 12)*.

A 12-lead ECG may show suggestive features. Intravenous calcium chloride is indicated in the presence of hyperkalaemia, hypocalcaemia, and calcium channel-blocker overdose.

Suspect **hypothermia** in any drowning incident *(chapter 12)*; use a low reading thermometer.

- **The four Ts**

A **tension pneumothorax** may be the primary cause of PEA and may follow attempts at central venous catheter insertion. The diagnosis is made clinically. Decompress rapidly by thoracostomy or needle thoracocentesis and then insert a chest drain.

Cardiac **tamponade** is difficult to diagnose because the typical signs of distended neck veins and hypotension cannot be assessed during cardiac arrest. Cardiac arrest after penetrating chest trauma or after cardiac surgery should raise strong suspicion of tamponade - the need for needle pericardiocentesis or resuscitative thoracotomy should be considered in this setting *(chapter 12)*.

In the absence of a specific history of accidental or deliberate ingestion, poisoning by therapeutic or toxic substances may be difficult to detect but in some cases may be revealed later by laboratory investigations *(chapter 12)*. Where available, the appropriate antidotes should be used but most often the required treatment is supportive.

The commonest cause of **thromboembolic** or mechanical circulatory obstruction is massive pulmonary embolism. If pulmonary embolism is thought to be the cause cardiac arrest consider giving a thrombolytic drug immediately. Following fibrinolysis during CPR for acute pulmonary embolism, survival and good neurological outcome have been reported in cases requiring in excess of 60 min of CPR. If a fibrinolytic drug is given in these circumstances, consider performing CPR for at least 60-90 min before termination of resuscitation attempts.

6.   Use of ultrasound during advanced life support

Although no studies have shown that use of this imaging modality improves outcome, there is no doubt that echocardiography has the potential to detect reversible causes of cardiac arrest. Specific protocols for ultrasound evaluation during CPR may help to identify potentially reversible causes (e.g. cardiac tamponade, pulmonary embolism, hypovolaemia, pneumothorax) and identify pseudo-PEA (organised myocardial contractions without palpable pulses). When available for use by trained clinicians, ultrasound may be of use in assisting with diagnosis and treatment of potentially reversible causes of cardiac arrest. The integration of ultrasound into advanced life support requires considerable training if interruptions to chest compressions are to be minimised. A sub-xiphoid probe position has been recommended. Placement of the probe just before chest compressions are paused for a planned rhythm assessment enables a well-trained operator to obtain views within 5 seconds. Absence of cardiac motion on sonography during resuscitation of patients in cardiac arrest is highly predictive of death although sensitivity and specificity has not been reported.

7.   Signs of life

If signs of life (such as regular respiratory effort, movement) or readings from patient monitors compatible with ROSC (e.g. sudden increase in exhaled carbon dioxide or arterial blood pressure waveform) appear during CPR, stop CPR briefly and check the monitor. If an organised rhythm is present, check for a pulse. If a pulse is palpable, continue post-resuscitation care and/or treatment of peri-arrest arrhythmias if appropriate. If no pulse is present, continue CPR.

The use of waveform capnography may enable ROSC to be detected without pausing chest compressions. A significant increase in $ETCO_2$ during CPR may be seen when ROSC occurs.

8. Waveform Capnography during advanced life support

8.1. Introduction

Carbon dioxide (CO_2) is a waste product of metabolism; approximately 400 L are produced each day. It is carried in the blood to the lungs where it is exhaled. The concentration in the blood is measured as the partial pressure of CO_2 (PCO_2) and in arterial blood ($PaCO_2$) is normally 5.3 kPa (4.7-6.0 kPa) = 40 mmHg (35-45 mmHg). The concentration of CO_2 can also be measured in expired air and is expressed as either percentage by volume or as a partial pressure, both of which are very similar numerically. The concentration varies throughout expiration, being maximal at the end and it is this value, the end-tidal CO_2 ($ETCO_2$) that is most useful. Figure 6.6 shows CO_2 curves during resuscitation starting with low quality chest compression, increase in CO_2 indicates good quality chest compressions with immediate sustained increase at ROSC.

Figure 6.6
Waveform capnography
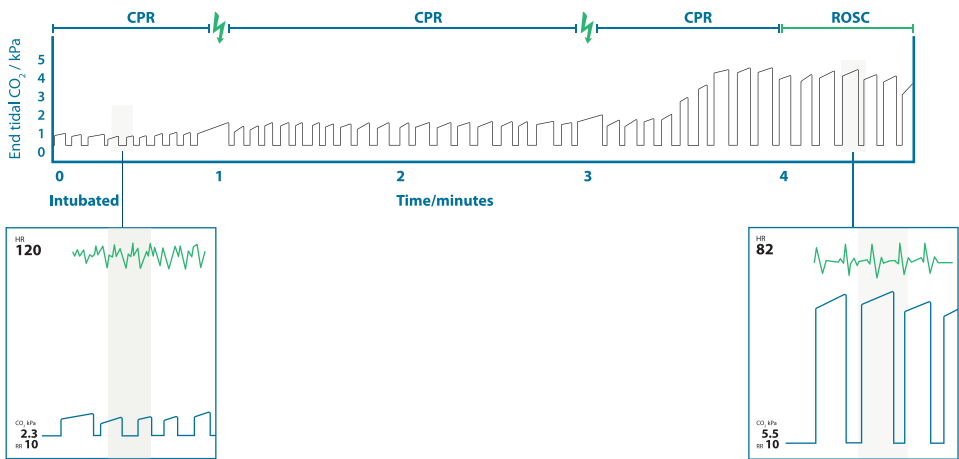
8.2. Nomenclature

The terms describing the measurement of carbon dioxide are derived from the Greek 'capnos', which means smoke. A capnometer is a device used to measure the concentration of CO_2 and gives a numerical value of the % or partial pressure (kPa) of the concentration of CO_2. A capnograph is a device that displays a waveform of the concentration of CO_2 as it varies during expiration and a numerical value. This is usually referred to as waveform capnography and is the most useful display for clinical use.

Figure 6.7
Waveform Capnography
"A: Start Expiration; B: End Expiration = ETCO$_2$ "
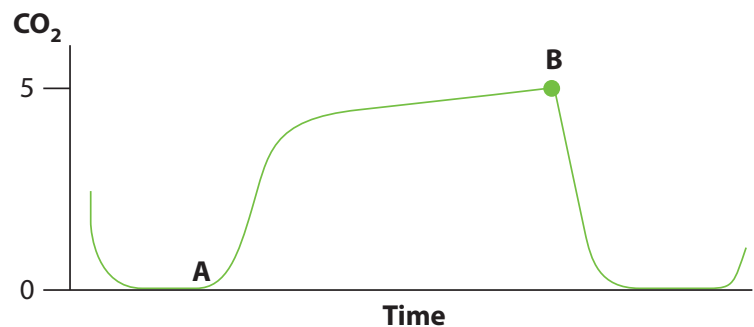
Figure 6.8
Spontaneous breathing
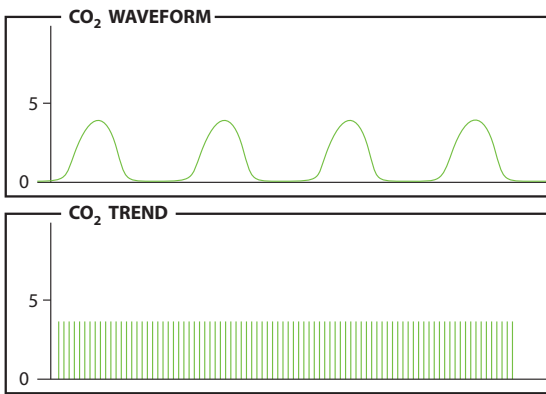

Figure 6.9
Ventilated patient
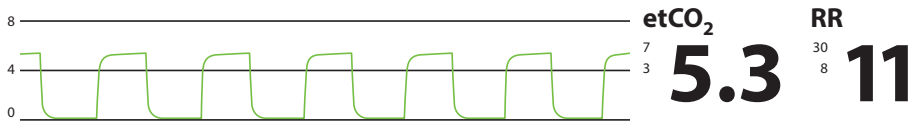
Figure 6.10
High-quality CPR
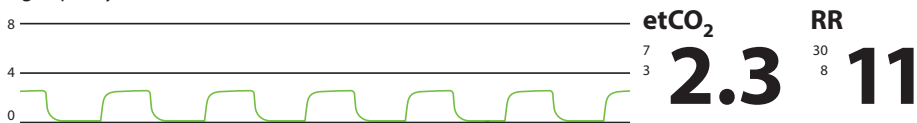
Figure 6.11
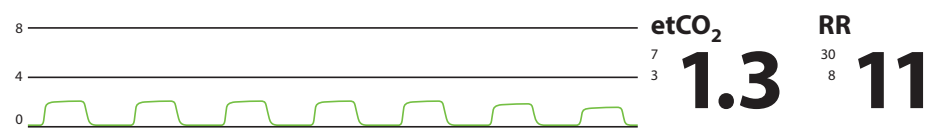Chest compression provider tiring

etCO$_2$ **1.3**   RR **11**

Figure 6.12
ETCO$_2$ with ROSC

etCO$_2$ **5.6**   RR **11**
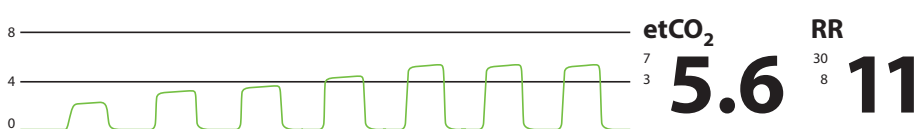
Figure 6.13
Persistently low ETCO$_2$ - associated with poor prognosis

etCO$_2$ **1.3**   RR **11**

Figure 6.14
Disconnection

etCO$_2$ **5**   RR **11**
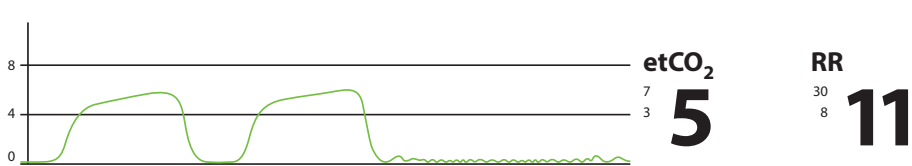
8.3. Equipment

In order to analyse the concentration of CO_2 in expired gas, most capnographs employ side-stream sampling. A connector (T-piece) is placed in the breathing system, usually on the end of the tracheal tube or supraglottic airway device (SAD). This has a small port on the side to which is attached a fine bore sampling tube. A continuous sample of gas is aspirated (about 50 ml min^{-1}) and analysed by using the property of absorption of infra-red light. The amount absorbed is proportional to the concentration of the absorbing molecule (in this case CO_2) and this is compared to a known standard, enabling the CO_2 concentration to be determined. An alternative system is main-stream sampling in which the infrared source and detector are contained within a cell or cuvette which is placed directly in the breathing system, usually between the tracheal tube or SAD and circuit. Gas is analysed as it passes through the sensor and none is removed from the system. Both systems are used in fixed or portable monitors.

- **What factors affect the end-tidal CO_2?**

There are 3 determinants of the ETCO$_2$:

- production by cellular metabolism

- transport to the lungs – the cardiac output

- elimination by ventilation

In health, the greatest variation is the result of changes in metabolism, usually increasing CO_2 production. This causes compensatory changes in transport, an increase in cardiac output, and elimination, an increase in ventilation to prevent accumulation of carbon dioxide.

In critically ill patients it is usually a failure in transportation (reduced cardiac output), elimination (inadequate ventilation) or a combination of both that causes changes in the arterial concentration of CO_2 and hence changes in the end-tidal CO_2. During a cardiac arrest, blood flow to the lungs ceases and despite continued production, if ventilation is maintained, ETCO$_2$ falls to zero. Once chest compressions are started, blood flow to the lungs will be partially restored and if the patient is ventilated, the end-tidal CO_2 will increase, proportionately to the cardiac output generated.

- **What information can be gained from monitoring ETCO$_2$ during cardiopulmonary resuscitation?**
    1. **Tube placement**
       Capnography has a high sensitivity and specificity for confirming placement of a tracheal tube in the airway.

    2. **Quality of CPR**
       The more efficient the chest compression, the greater the cardiac output which delivers more CO_2 to the lungs from where it is exhaled thus generating a higher end-tidal concentration. High-quality chest compressions will result in typical ETCO$_2$ values of 2.0-2.5 kPa.

3. Return of Spontaneous Circulation (ROSC)

With ROSC, there is an immediate, sustained increase in $ETCO_2$. This is often the first indicator of ROSC. This often precedes other indicators such as the presence of a palpable pulse. It is a result of the circulation transporting accumulated carbon dioxide from the tissues to the lungs and often results in an initial raised $ETCO_2$.

4. Guide to rate of ventilation

Hyperventilation of the patient's lungs by rescuers is common during cardiac arrest, and usually the result of increased rate rather than tidal volume. Excessive ventilation reduces coronary perfusion and survival. Waveform capnography provides a measure of ventilation rate during CPR and may therefore reduce the incidence of hyperventilation.

5. Prognostication

A higher $ETCO_2$ during resuscitation is associated with an increased likelihood of ROSC and chance of survival to discharge. In one study, an $ETCO_2$ of < 1.9 kPa (14 mmHg) during resuscitation had a sensitivity and specificity of 100 % in predicting non-survivors. After cardiac arrest and CPR for more than 30 minutes, exhaled carbon dioxide values decrease and may become zero. The inter-individual differences and influence of cause of cardiac arrest, the problem with self-fulfilling prophecy in studies, our lack of confidence in the accuracy of measurement during CPR, and the need for an advanced airway to measure end-tidal CO_2 reliably limits our confidence in its use for prognostication. Thus, we recommend that a specific end-tidal CO_2 value at any time during CPR should not be used alone to stop CPR efforts. End-tidal CO_2 values should be considered only as part of a multi-modal approach to decision-making for prognostication during CPR.

6

9. Discontinuing resuscitation and diagnosing death

If attempts at obtaining ROSC are unsuccessful the cardiac arrest team leader should discuss stopping CPR with the resuscitation team. The decision to stop CPR requires clinical judgement and a careful assessment of the likelihood of achieving ROSC.

After stopping CPR, observe the patient for a minimum of 5 min before confirming death. The absence of mechanical cardiac function is normally confirmed using a combination of the following:

- absence of a central pulse on palpation

- absence of heart sounds on auscultation

One or more of the following can supplement these criteria:

- asystole on a continuous ECG display

- absence of pulsatile flow using direct intra-arterial pressure monitoring

- absence of contractile activity using echocardiography

Any return of cardiac or respiratory activity during this period of observation should prompt a further 5 min observation from the next point of cardiorespiratory arrest. After 5 min of continued cardiorespiratory arrest, the absence of the pupillary responses to light, of the corneal reflexes, and of any motor response to supra-orbital pressure should be confirmed. The time of death is recorded as the time at which these criteria are fulfilled.

FURTHER READING

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6

Chapter 7.

Airway management and ventilation

Section 1
Basic airway management and ventilation

LEARNING OUTCOMES

To understand:

- the causes and recognition of airway obstruction
- techniques for airway management when starting resuscitation
- the use of simple adjuncts to maintain airway patency
- the use of simple devices for ventilating the lungs

1.   Introduction

Patients requiring resuscitation often have an obstructed airway, usually caused by loss of consciousness, but occasionally it may be the primary cause of cardiorespiratory arrest. Prompt assessment, with control of airway patency and provision of ventilation if required are essential. This will help to prevent secondary hypoxic damage to the brain and other vital organs. Without adequate oxygenation it may be impossible to restore an organised, perfusing cardiac rhythm. These principles may not apply to the witnessed primary cardiac arrest in the vicinity of a defibrillator; in this case, the priority is immediate defibrillation followed by attention to the airway.

2.   Causes of airway obstruction

Obstruction of the airway may be partial or complete. It may occur at any level, from the nose and mouth down to the trachea. In the unconscious patient, the commonest site of airway obstruction is at the soft palate and epiglottis. Obstruction may also be caused by vomit or blood (regurgitation of gastric contents or trauma), or by foreign bodies. Laryngeal obstruction may be caused by oedema from burns, inflammation or anaphylaxis.

Upper airway stimulation may cause laryngeal spasm. Obstruction of the airway below the larynx is less common, but may arise from excessive bronchial secretions, mucosal oedema, bronchospasm, pulmonary oedema or aspiration of gastric contents.

3. Recognition of airway obstruction

Airway obstruction can be subtle and is often missed by healthcare professionals, let alone by laypeople. The 'look, listen and feel' approach is a simple, systematic method of detecting airway obstruction.

- LOOK for chest and abdominal movements.
- LISTEN and FEEL for airflow at the mouth and nose.

In partial airway obstruction, air entry is diminished and usually noisy:

- Inspiratory stridor - caused by obstruction at the laryngeal level or above.
- Expiratory wheeze - suggests obstruction of the lower airways, which tend to collapse and obstruct during expiration.
- Gurgling - suggests the presence of liquid or semisolid foreign material in the upper airways.
- Snoring - arises when the pharynx is partially occluded by the tongue or palate.
- Crowing or stridor - is the sound of laryngeal spasm or obstruction.

Complete airway obstruction in a patient who is making respiratory efforts causes paradoxical chest and abdominal movement, described as 'see-saw breathing'.

During airway obstruction, accessory muscles of respiration are used – the neck and the shoulder muscles contract to assist movement of the thoracic cage. There may also be intercostal and subcostal recession and a tracheal tug. Full examination of the neck, chest and abdomen should enable differentiation of the movements associated with complete airway obstruction from those of normal breathing. Listen for airflow: normal breathing should be quiet, completely obstructed breathing will be silent, and noisy breathing indicates partial airway obstruction. During apnoea, when spontaneous breathing movements are absent, complete airway obstruction is recognised by failure to inflate the lungs during attempted positive pressure ventilation.

3.1. Patients with tracheostomies or permanent tracheal stomas

A patient with a tracheostomy tube or a permanent tracheal stoma (usually following a laryngectomy) may develop airway obstruction from blockage of the tracheostomy tube or stoma - airway obstruction cannot occur at the level of the pharynx in these patients. Remove any obvious foreign material from the stoma or tracheostomy tube. If necessary, remove the tracheostomy tube or, if present, exchange the tracheostomy tube liner. If a blocked tracheostomy tube is removed it should be possible to ventilate the patient's lungs by sealing the stoma and using a bag-mask applied to the face, or by intubating the trachea

orally with a standard tracheal tube. In a patient with a permanent tracheal stoma, give oxygen and, if required, assist ventilation via the stoma, and not the mouth.

3.2. Choking

Foreign body airway obstruction (FBAO) is an uncommon but potentially treatable cause of accidental death. As most choking events are associated with eating, they are commonly witnessed. As victims initially are conscious and responsive, there are often opportunities for early interventions which can be life saving.

3.2.1. Recognition

Foreign bodies may cause either mild or severe airway obstruction. The signs and symptoms enabling differentiation between mild and severe airway obstruction are summarised in table 7.1.

3.2.2. Sequence for the treatment of adult choking

1. If the patient shows signs of mild airway obstruction *(figure 7.1)*:

    •	Encourage him to continue coughing, but do nothing else.

2. If the patient shows signs of severe airway obstruction and is conscious:

    •	Give up to 5 back blows:

        -	Stand to the side and slightly behind the patient.

        -	Support the chest with one hand and lean the patient well forwards.

        -	Give up to 5 sharp blows between the scapulae with the heel of the other hand.

    •	Check to see if each back blow has relieved the airway obstruction.

    •	If 5 back blows fail to relieve the airway obstruction give up to 5 abdominal thrusts.

        -	Stand behind the patient and put both arms round the upper part of his abdomen.

        -	Place a clenched fist just under the xiphisternum; grasp this hand with your other hand and pull sharply inwards and upwards.

        -	Repeat up to 5 times.

    •	If the obstruction is still not relieved, continue alternating 5 back blows with 5 abdominal thrusts.

3. If the patient becomes unconscious, call the resuscitation team and start CPR.

4. As soon as an individual with appropriate skills is present, undertake laryngoscopy and attempt to remove any foreign body with Magill's forceps.

Table 7.1
Signs of choking

General signs of choking:	
• Attack occurs while eating.	
• Patient may clutch his neck.	
Signs of severe airway obstruction:	**Signs of mild airway obstruction:**
Response to question 'Are you choking?'	*Response to question 'Are you choking?'*
• Patient is unable to speak.	• Patient speaks and answers yes.
• Patient may respond by nodding.	
Other signs:	*Other signs:*
• Patient is unable to breathe.	• Patient is able to speak, cough, and breathe.
• Breathing sounds wheezy.	
• Attempts at coughing are silent.	
• Patient may be unconscious.	

Figure 7.1
Adult choking algorithm
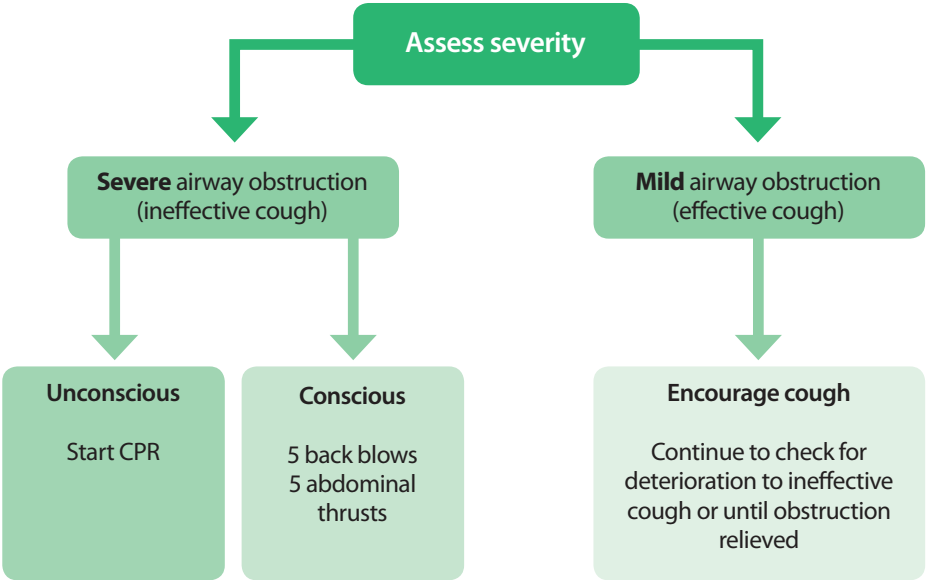
4. Basic techniques for opening the airway

Once airway obstruction is recognised, take immediate action to relieve the obstruction and maintain a clear airway. Three manoeuvres that can be used to relieve upper airway obstruction are:

- head tilt
- chin lift
- jaw-thrust

4.1. Head tilt and chin lift

Place one hand on the patient's forehead and tilt the head back gently; place the fingertips of the other hand under the point of the patient's chin, and gently lift to stretch the anterior neck structures *(figure 7.2)*.

Figure 7.2
Head tilt and chin lift

4.2. Jaw-thrust

Jaw-thrust is an alternative manoeuvre for bringing the mandible forward and relieving obstruction by the tongue, soft palate and epiglottis *(figure 7.3)*. It is most successful when applied with a head tilt.

4.2.1. Procedure for jaw-thrust

- Identify the angle of the mandible.
- With the index and other fingers placed behind the angle of the mandible, apply steady upwards and forward pressure to lift the mandible.
- Using the thumbs, slightly open the mouth by downward displacement of the chin.

These simple positional methods are successful in most cases where airway obstruction is caused by loss of muscle tone in the pharynx. After each manoeuvre, check for success using the look, listen and feel sequence. If a clear airway cannot be achieved, look for other causes of airway obstruction. Use a finger sweep to remove any solid foreign material visible in the mouth. Remove broken or displaced dentures but leave well-fitting dentures in place as they help to maintain the contours of the mouth, facilitating a good seal for ventilation by mouth-to-mask or bag-mask techniques.

Figure 7.3
Jaw-thrust

4.3. Airway manoeuvres in a patient with suspected cervical spine injury

If spinal injury is suspected (e.g. if the victim has fallen, been struck on the head or neck, or has been rescued after diving into shallow water) maintain the head, neck, chest, and lumbar region in the neutral position during resuscitation. Excessive head tilt could aggravate the injury and damage the cervical spinal cord; however, this complication remains theoretical and the relative risk is unknown. When there is a risk of cervical spine injury, establish a clear upper airway by using jaw-thrust or chin lift in combination with manual in-line stabilisation (MILS) of the head and neck by an assistant. If life-threatening airway obstruction persists despite effective application of jaw-thrust or chin lift, add head tilt a small amount at a time until the airway is open; establishing a patent airway takes priority over concerns about a potential cervical spine injury.

5.   Adjuncts to basic airway techniques

Despite a total lack of published data on the use of nasopharyngeal and oropharyngeal airways during CPR, they are often helpful, and sometimes essential, to maintain an open airway, particularly when resuscitation is prolonged. The position of the head and neck is maintained to keep the airway aligned. Oropharyngeal and nasopharyngeal airways overcome backward displacement of the soft palate and tongue in an unconscious patient, but head tilt and jaw-thrust may also be required.

5.1.   Oropharyngeal airway

Oropharyngeal airways are available in sizes suitable for the newborn to large adults. An estimate of the size required is obtained by selecting an airway with a length corresponding to the vertical distance between the patient's incisors and the angle of the jaw. The most common sizes are 2, 3 and 4 for small, medium and large adults, respectively. During insertion of an oropharyngeal airway, the tongue can occasionally be pushed backwards, exacerbating obstruction instead of relieving it. The oropharyngeal airway may lodge in the vallecula, or the epiglottis may obstruct the lumen. Ensuring a correct insertion technique should avoid this problem. Attempt insertion only in unconscious patients: vomiting or laryngospasm may occur if glossopharyngeal or laryngeal reflexes are present.

5.1.1. Technique for insertion of an oropharyngeal airway:

- Open the patient's mouth and ensure that there is no foreign material that may be pushed into the larynx (if there is any, then use suction to remove it).

- Insert the airway into the oral cavity in the 'upside-down' position as far as the junction between the hard and soft palate and then rotate it through 180° *(figure 7.5)*. Advance the airway until it lies within the pharynx. This rotation technique minimises the chance of pushing the tongue backwards and downwards. Remove the airway if the patient gags or strains. Correct placement is indicated by an improvement in airway patency and by the seating of the flattened reinforced section between the patient's teeth or gums (if edentulous). A jaw-thrust may further aid final placement of the airway as it is finally pushed into the correct position.

After insertion, maintain head-tilt/chin-lift or jaw-thrust, and check the patency of the airway and ventilation using the look, listen and feel technique. Where there is suspicion of an injury to the cervical spine, maintain alignment and immobilisation of the head and neck. Suction is usually possible through an oropharyngeal airway using a fine bore flexible suction catheter.

Figure 7.4
Sizing an oropharyngeal airway

Figure 7.5
Oropharyngeal airway insertion

5.2. Nasopharyngeal airway

In patients who are not deeply unconscious, a nasopharyngeal airway is tolerated better than an oropharyngeal airway. The nasopharyngeal airway may be life saving in patients with clenched jaws, trismus or maxillofacial injuries, when insertion of an oral airway is impossible.

Inadvertent insertion of a nasopharyngeal airway through a fracture of the skull base and into the cranial vault is possible, but extremely rare. In the presence of a known or suspected basal skull fracture an oral airway is preferred, but if this is not possible, and the

airway is obstructed, gentle insertion of a nasopharyngeal airway may be life-saving (i.e. the benefits may far outweigh the risks).

The tubes are sized in millimetres according to their internal diameter, and the length increases with diameter. The traditional methods of sizing a nasopharyngeal airway (measurement against the patient's little finger or anterior nares) do not correlate with the airway anatomy and are unreliable. Sizes 6-7 mm are suitable for adults. Insertion can cause damage to the mucosal lining of the nasal airway, resulting in bleeding in up to 30 % of cases. If the tube is too long it may stimulate the laryngeal or glossopharyngeal reflexes to produce laryngospasm or vomiting.

5.2.1. Technique for insertion of a nasopharyngeal airway

- Check for patency of the right nostril.

- Some designs require a safety pin to be inserted through the flange to provide an extra precaution against the airway disappearing beyond the nares. The safety pin should be inserted BEFORE inserting the airway.

- Lubricate the airway thoroughly using water-soluble gel.

- Insert the airway bevel end first, vertically along the floor of the nose with a slight twisting action *(figure 7.6)*. The curve of the airway should direct it towards the patient's feet. If any obstruction is met, remove the tube and try the left nostril.

- Once in place, use the look, listen and feel technique to check the patency of the airway and adequacy of ventilation. Chin lift or jaw-thrust may still be required to maintain airway patency. Where there is suspicion of an injury to the cervical spine, maintain correct alignment and immobilisation of the head and neck.

Figure 7.6
Nasopharyngeal airway insertion

6. Oxygen

During CPR, give the maximal feasible inspired oxygen concentration. A self-inflating bag can be connected to a facemask, tracheal tube or supraglottic airway (SGA). Without supplementary oxygen, the self-inflating bag ventilates the patient's lungs with ambient air (21 % oxygen). The delivered oxygen concentration can be increased to about 85 % by using a reservoir system and attaching oxygen at a flow 15 l min^{-1}. After ROSC, as soon as arterial blood oxygen saturation can be monitored reliably (by blood gas analysis and/or pulse oximetry), titrate the inspired oxygen concentration to maintain the arterial blood oxygen saturation in the range of 94-98 %. Avoid hypoxaemia, which is also harmful – ensure reliable measurement of arterial oxygen saturation before reducing the inspired oxygen concentration.

7. Ventilation

Artificial ventilation is started as soon as possible in any patient in whom spontaneous ventilation is inadequate or absent. Expired air ventilation (rescue breathing) is effective but the rescuer's expired oxygen concentration is only 16-17 %; so it must be replaced as soon as possible by ventilation with oxygen-enriched air. Although mouth-to-mouth ventilation has the benefit of not requiring any equipment, the technique is aesthetically unpleasant, particularly when vomit or blood is present, and the rescuer may be reluctant to place themselves in intimate contact with the victim who may be unknown to them.

There are only isolated reports of individuals acquiring infections after providing CPR, e.g. tuberculosis and severe acute respiratory distress syndrome (SARS). Transmission of HIV during provision of CPR has never been reported. Simple adjuncts are available to enable direct person-to-person contact to be avoided; some of these devices may reduce the risk of cross infection between patient and rescuer.

The pocket resuscitation mask is similar to an anaesthetic facemask, and enables mouth-to-mask ventilation. It has a unidirectional valve, which directs the patient's expired air away from the rescuer. The mask is transparent so that vomit or blood from the patient can be seen. Some masks have a connector for the addition of oxygen. When using masks without a connector, supplemental oxygen can be given by placing the tubing underneath one side and ensuring an adequate seal. Use a two-hand technique to maximise the seal with the patient's face.

High airway pressures can be generated if the tidal volume or inspiratory flow is excessive, predisposing to gastric inflation and subsequent risk of regurgitation and pulmonary aspiration. The risk of gastric inflation is increased by:

- malalignment of the head and neck, and an obstructed airway

- an incompetent oesophageal sphincter (present in all patients with cardiac arrest)

- a high airway inflation pressure

Conversely, if inspiratory flow is too low, inspiratory time will be prolonged and the time available to give chest compressions is reduced. Deliver each breath over approximately 1 second, giving a volume that corresponds to normal chest movement; this represents a compromise between giving an adequate volume, minimising the risk of gastric inflation, and allowing adequate time for chest compressions. During CPR with an unprotected airway, give two ventilations after each sequence of 30 chest compressions.

Inadvertent hyperventilation during CPR is common. While this increased intrathoracic pressure and peak airway pressures in small case series in humans, a carefully controlled animal experiment revealed no adverse effects. During continuous chest compressions a ventilation rate of 10 min^{-1} with an advanced airway is recommended.

7.1. Technique for mouth-to-mask ventilation

- Place the patient supine with the head in a 'sniffing' position i.e. neck slightly flexed on a pillow with the head extended (tilted backwards) on the neck.

- Apply the mask to the patient's face using the thumbs of both hands.

- Lift the jaw into the mask with the remaining fingers by exerting pressure behind the angles of the jaw (jaw-thrust). At the same time, press the mask onto the face with the thumbs to make a tight seal *(figure 7.7)*.

- Blow gently through the inspiratory valve and watch the chest rise normally.

- Stop inflation and observe the chest falling.

- Any leaks between the face and mask can be reduced by adjusting the contact pressure, altering the position of the fingers and thumbs, or increasing jaw-thrust.

- If oxygen is available, add it via the port at a flow 15 l min^{-1}.

7.2. Self-inflating bag

The self-inflating bag can be connected to a face mask, tracheal tube, or supraglottic airway device. As the bag is squeezed, the contents are delivered to the patient's lungs. On release, the expired gas is diverted to the atmosphere via a one-way valve; the bag then refills automatically via an inlet at the opposite end. When used without supplemental oxygen, the self-inflating bag ventilates the patient's lungs with ambient air (oxygen concentration 21 %). This is increased to around 45 % by attaching high-flow oxygen directly to the bag adjacent to the air intake. An inspired oxygen concentration of approximately 85 % is achieved if a reservoir system is attached and the oxygen flow is maximally increased. As the bag re-expands it fills with oxygen from both the reservoir and the continuous flow from the attached oxygen tubing. Using demand valves with a bag enables oxygen concentrations close to 100 %.

Although the bag-mask apparatus enables ventilation with high concentrations of oxygen, its use by a single person requires considerable skill. When used with a face mask, it is often difficult to achieve a gas-tight seal between the mask and the patient's face, and maintain a patent airway with one hand whilst squeezing the bag with the other. Any significant leak will cause hypoventilation and if the airway is not patent, gas may also be forced into the

stomach. This will reduce ventilation further and greatly increase the risk of regurgitation and aspiration. There is a natural tendency to try to compensate for a leak by excessive compression of the bag, which causes high peak airway pressures and forces more gas into the stomach. Some self-inflating bags have flow restrictors that limit peak airway pressure with the aim of reducing gastric inflation.

The two-person technique for bag-mask ventilation is preferable *(figure 7.7)*. One person holds the face mask in place using a jaw-thrust with both hands and an assistant squeezes the bag. In this way, a better seal can be achieved and the patient's lungs can be ventilated more effectively and safely.

Figure 7.7
The two-person technique for bag-mask ventilation

KEY LEARNING POINTS

- Airway patency and ventilating the lungs are important components of CPR.

- Use of simple airway manoeuvres, with or without basic adjuncts, will often achieve a patent airway.

- Give all patients high-concentration oxygen until the arterial oxygen saturation is measurable.

Section 2
Alternative airway devices

LEARNING OUTCOMES

To understand:

- the role of supraglottic airway devices during CPR

1.　Introduction

Effective bag-mask ventilation requires a reasonable level of skill and experience: the inexperienced are likely to achieve ineffective tidal volumes and cause gastric inflation with risk of regurgitation and pulmonary aspiration. The tracheal tube has generally been considered the optimal method of managing the airway during cardiac arrest. There is evidence that, without adequate training and experience, the incidence of complications, such as unrecognised oesophageal intubation (2.4-17 % in several studies involving paramedics) and dislodgement, is unacceptably high. Prolonged attempts at tracheal intubation are harmful; the cessation of chest compressions during this time will compromise coronary and cerebral perfusion. Several alternative airway devices have been used for airway management during CPR. In comparison with bag-mask ventilation, use of supraglottic airway devices (SGAs) may enable more effective ventilation and reduce the risk of gastric inflation. Furthermore, SGAs are easier to insert than a tracheal tube.

There are no data supporting the routine use of any specific approach to airway management during cardiac arrest. The best technique is dependent on the precise circumstances of the cardiac arrest and the competence of the rescuer.

2.　Laryngeal mask airway

The laryngeal mask airway (LMA) consists of a wide-bore tube with an elliptical inflated cuff designed to seal around the laryngeal opening. The original LMA (classic LMA [cLMA]), which is reusable, has been studied during CPR, but none of these studies has compared it directly with the tracheal tube. Although the cLMA remains in common use in elective anaesthetic practice, it has been superseded by several 2nd generation SGAs that have more favourable characteristics, particularly when used for emergency airway management. Most of these SGAs are single use and achieve higher oropharyngeal seal pressures than the cLMA, and some incorporate gastric drain tubes.

2.1.   Technique for insertion of a laryngeal mask airway

- Maintain chest compressions throughout the insertion attempt.

- Select a LMA of an appropriate size for the patient and deflate the cuff fully. A size 5 will be correct for most men and a size 4 for most women. Lubricate the outer face of the cuff area (the part that will not be in contact with the larynx) with water-soluble gel.

- Flex the patient's neck slightly and extend the head (try to maintain neutral alignment of the head and neck if there is suspicion of cervical spine injury).

- Holding the LMA like a pen, insert it into the mouth *(figure 7.8)*. Advance the tip behind the upper incisors with the upper surface applied to the palate until it reaches the posterior pharyngeal wall. Press the mask backwards and downwards around the corner of the pharynx until a resistance is felt as it locates in the back of the pharynx. If possible, get an assistant to apply a jaw-thrust after the LMA has been inserted into the mouth - this increases the space in the posterior pharynx and makes successful placement easier. A slight 45 degree twist will often aid placement if initial attempts at insertion beyond the pharynx are proving difficult.

- Connect the inflating syringe and inflate the cuff with air (40 ml for a size 5 LMA and 30 ml for a size 4 LMA); alternatively, inflate the cuff to a pressure of 60 cmH$_2$O. If insertion is satisfactory, the tube will lift 1-2 cm out of the mouth as the cuff finds its correct position and the larynx is pushed forward.

- If the LMA has not been inserted successfully after 30 s, oxygenate the patient using a pocket mask or bag-mask before reattempting LMA insertion.

- Confirm a clear airway by listening over the chest during inflation and observing bilateral chest movement. A large, audible leak suggests malposition of the LMA, but a small leak is acceptable provided chest rise is adequate.

- Insert a bite block alongside the tube if available and secure the LMA with a bandage or tape.

Figure 7.8
Insertion of a laryngeal mask airway

3. i-gel airway

The i-gel is also a supraglottic airway. The cuff is made of thermoplastic elastomer gel and does not require inflation; the stem of the i-gel incorporates a bite block and a narrow oesophageal drain tube. It is easy to insert, requiring only minimal training and a laryngeal seal pressure of 20-24 cmH$_2$O can be achieved. In two manikin studies, insertion of the i-gel was significantly faster than several other airway devices. The ease of insertion of the i-gel and its favourable leak pressure make it theoretically very attractive as a resuscitation airway device for those inexperienced in tracheal intubation. Use of the i-gel during cardiac arrest has been reported but more data on its use in this setting are awaited.

4. Laryngeal tube

The laryngeal tube (LT) is another supraglottic airway device commonly used in the anaesthetic setting and out of hospital. It is a single-lumen tube with both an oesophageal and pharyngeal cuff. A single pilot balloon inflates both cuffs simultaneously and it is available in a variety of sizes. Successful insertion and airway pressures generated are comparable to the LMA when performed by non-anaesthetists. A double lumen LT with an oesophageal vent, an intubation LT and a disposable version (LT-D) are available.

4.1. Technique for insertion of a laryngeal tube airway

- Maintain chest compressions throughout the insertion attempt. Select a LT of an appropriate size for the patient and deflate the cuff fully. A size 5 will be correct when the patient's height is > 180 cm; size 4 when 155-180 cm; and a size 3 when < 155 cm. Lubricate the tip of the LT with water-soluble gel.

- Place the patient's head and neck in the sniffing or neutral position (try to maintain neutral alignment of the head and neck if there is suspicion of cervical spine injury).

- The tip of the LT should be placed against the hard palate below the incisors. Slide the LT down the centre of the mouth until resistance is felt or the device is almost fully inserted. When the LT is inserted properly, the second bold black line on the tube should have just passed between upper and lower teeth.

- Inflate the cuff to a pressure of 60 cmH$_2$O. This can be done either with a cuff inflator or a 100 ml syringe with the marks for the recommended volumes for each size of the LT.

- If the LT has not been inserted successfully after 30 sec, oxygenate the patient using a pocket mask or bag-mask before reattempting LT insertion.

- Confirm a clear airway by listening over the chest during inflation and observing bilateral chest movement. A large, audible leak suggests malposition of the LT, but a small leak is acceptable provided chest rise is adequate.

- Insert a bite block alongside the tube if available and secure the LT with a bandage or tape.

5. Limitations of all SGAs

- In the presence of high airway resistance or poor lung compliance (pulmonary oedema, bronchospasm, chronic obstructive pulmonary disease) there is a risk of a significant leak around the cuff causing hypoventilation. Most of the gas leaking around the cuff normally escapes through the patient's mouth but some gastric inflation may occur.

- There are no data demonstrating whether or not it is possible to provide adequate ventilation via an SGA without interruption of chest compressions. Uninterrupted chest compressions are likely to cause at least some gas leak around the SGA cuff when ventilation is attempted. Attempt continuous compressions initially but abandon this if persistent leaks and hypoventilation occur.

- There is a theoretical risk of aspiration of stomach contents because the SGA does not sit within the larynx like a tracheal tube; however, this complication has not been documented widely in clinical practice.

- If the patient is not deeply unconscious, insertion of the SGA may cause coughing, straining or laryngeal spasm. This will not occur in patients in cardiorespiratory arrest.

- If an adequate airway is not achieved withdraw the SGA and attempt reinsertion ensuring a good alignment of the head and neck.

- Uncommonly, airway obstruction may be caused by pushing the tongue towards the posterior pharynx. Withdraw the SGA and attempt reinsertion.

To become proficient in the insertion of any SGA requires practice on patients and this should be achieved under the supervision of an appropriately experienced person (e.g. anaesthetist) in a controlled environment.

KEY LEARNING POINTS

- Supraglottic airway devices are good alternatives to bag-mask devices and can be used instead of the bag-mask technique.

- Supraglottic airway devices should be used instead of tracheal intubation unless individuals highly skilled in intubation are immediately available. They should also be used if attempted intubation is unsuccessful.

Section 3
Tracheal intubation and cricothyroidotomy

LEARNING OUTCOMES

To understand:

- the advantages and disadvantages of tracheal intubation during cardiopulmonary resuscitation
- some methods for confirming correct placement of a tracheal tube
- the role of needle and surgical cricothyroidotomy

1. Tracheal intubation

There is insufficient evidence to support or refute the use of any specific technique to maintain an airway and provide ventilation in adults with cardiorespiratory arrest. Despite this, tracheal intubation is perceived as the optimal method of providing and maintaining a clear and secure airway. It should be used only when trained personnel are available to carry out the procedure with a high level of skill and competence.

The perceived advantages of tracheal intubation over bag-mask ventilation include:

- enabling ventilation without interrupting chest compressions
- enabling effective ventilation, particularly when lung and/or chest compliance is poor
- minimising gastric inflation and therefore the risk of regurgitation
- protection against pulmonary aspiration of gastric contents
- the potential to free the rescuer's hands for other tasks

The perceived disadvantages of tracheal intubation over bag-valve-mask ventilation include:

- The risk of an unrecognised misplaced tracheal tube.
- A prolonged period without chest compressions while intubation is attempted - Tracheal intubation attempts accounted for almost 25 % of all CPR interruptions.
- A comparatively high failure rate.
- Tracheal intubation is a difficult skill to acquire and maintain.

Healthcare personnel who undertake prehospital intubation should do so only within a structured, monitored programme, which should include comprehensive competency-based training and regular opportunities to refresh skills. Rescuers must weigh the risks and benefits of intubation against the need to provide effective chest compressions. The intubation attempt may require some interruption of chest compressions but, once an advanced airway is in place, ventilation will not require interruption of chest compressions. Personnel skilled in advanced airway management should be able to undertake laryngoscopy without stopping chest compressions; a brief pause in chest compressions will be required only as the tube is passed through the vocal cords. Alternatively, to avoid any interruptions in chest compressions, the intubation attempt may be deferred until ROSC. The intubation attempt should interrupt chest compressions for less than 5 seconds; if intubation is not achievable within these constraints, recommence bag-mask ventilation. After intubation, tube placement must be confirmed and the tube secured adequately.

1.1. Confirmation of correct tracheal tube placement

Unrecognised oesophageal intubation is the most serious complication of attempted tracheal intubation. Routine use of primary and secondary techniques to confirm correct placement of the tracheal tube should reduce this risk.

1.1.1. Clinical assessment

Primary assessment includes observation of chest expansion bilaterally, auscultation over the lung fields bilaterally in the axillae (breath sounds should be equal and adequate) and over the epigastrium (breath sounds should not be heard). Clinical signs of correct tube placement (condensation in the tube, chest rise, breath sounds on auscultation of lungs, and inability to hear gas entering the stomach) are not reliable.

Confirmation of tracheal tube placement by an exhaled carbon dioxide or oesophageal detection device should reduce the risk of unrecognised oesophageal intubation but the performance of the available devices varies considerably. Furthermore, none of the these techniques will differentiate between a tube placed in a main bronchus and one placed correctly in the trachea.

1.1.2. Oesophageal detector device

The oesophageal detector device creates a suction force at the tracheal end of the tracheal tube, either by pulling back the plunger on a large syringe or releasing a compressed flexible bulb. Air is aspirated easily from the lower airways through a tracheal tube placed in the cartilage-supported rigid trachea. When the tube is in the oesophagus, air cannot be aspirated because the oesophagus collapses when aspiration is attempted. The oesophageal detector device may be misleading in patients with morbid obesity, late pregnancy or severe asthma or when there are copious tracheal secretions; in these conditions the trachea may collapse when aspiration is attempted.

1.1.3. **Carbon dioxide detectors**

Carbon dioxide (CO_2) detector devices measure the concentration of exhaled carbon dioxide from the lungs. The persistence of exhaled CO_2 after six ventilations indicates placement of the tracheal tube in the trachea or a main bronchus.

Broadly, there are three types of carbon dioxide detector devices:

1. Disposable colorimetric end-tidal carbon dioxide ($ETCO_2$) detectors use a litmus paper to detect CO_2, and these devices generally give readings of purple ($ETCO_2 < 0.5\%$), tan ($ETCO_2$ 0.5-2%) and yellow ($ETCO_2 > 2\%$).
   Tracheal placement of the tube is considered verified if the tan colour persists after a few ventilations. Although colorimetric CO_2 detectors identify placement quite well in patients with good perfusion, these devices are less accurate than clinical assessment in cardiac arrest patients because pulmonary blood flow may be so low that there is insufficient exhaled carbon dioxide. Furthermore, if the tracheal tube is in the oesophagus, six ventilations may lead to gastric distension, vomiting and aspiration.

2. Non-waveform electronic digital $ETCO_2$ devices generally measure $ETCO_2$ using an infrared spectrometer and display the results with a number; they do not provide a waveform graphical display of the respiratory cycle on a capnograph.

3. End-tidal CO_2 detectors that include a waveform graphical display (capnograph) are the most reliable for verification of tracheal tube position during cardiac arrest. Studies of waveform capnography to verify tracheal tube position in victims of cardiac arrest demonstrate 100% sensitivity and 100% specificity in identifying correct tracheal tube placement.

Waveform capnography is the most sensitive and specific way to confirm and continuously monitor the position of a tracheal tube in victims of cardiac arrest and should supplement clinical assessment (auscultation and visualisation of tube through cords). Waveform capnography will not discriminate between tracheal and bronchial placement of the tube - careful auscultation is essential. Existing portable monitors make capnographic initial confirmation and continuous monitoring of tracheal tube position feasible in almost all settings, including out-of-hospital, emergency department, and in-hospital locations where tracheal intubation is performed. Furthermore, waveform capnography may be a sensitive indicator of ROSC. Such waveform analysis may prove useful in PEA cardiac arrests.

2. Cricothyroidotomy

Occasionally it will be impossible to ventilate an apnoeic patient with a bag-mask, or to pass a tracheal tube or other airway device. This may occur in patients with extensive facial trauma or laryngeal obstruction caused by oedema, e.g. anaphylaxis, or foreign material. In these circumstances, it will be necessary to create a surgical airway below the level of the obstruction.

7

Surgical cricothyroidotomy provides a definitive airway that can be used to ventilate the patient's lungs until semi-elective intubation or tracheostomy is performed. Needle cricothyroidotomy is a much more temporary procedure providing only short-term oxygenation. It requires a wide-bore, non-kinking cannula, a high-pressure oxygen source and may cause serious barotrauma. It is also prone to failure because of kinking of the cannula, and is unsuitable for patient transfer.

2.1. Surgical cricothyroidotomy

Unlike needle cricothyroidotomy, the surgical technique will result in an airway that is protected by a cuffed tube. Higher airway pressures can be generated and tracheal suction is possible. Surgical cricothyroidotomy enables ventilation of the lungs despite complete airway obstruction at, or above, the glottis.

2.1.1. Procedure for surgical cricothyroidotomy

- Place the patient supine with the head extended if possible.

- Identify the cricothyroid membrane as the recess just above the cricoid cartilage and below the thyroid cartilage.

- Incise the skin over the membrane and extend the incision through the cricothyroid membrane. Make a vertical incision in the skin and a horizontal one into the cricothyroid membrane; this avoids the superiorly positioned cricothyroid artery.

- Use the reversed handle of a scalpel or tissue expanding forceps to open up the incision in the cricothyroid membrane.

- Insert a suitably sized tracheal tube into the trachea and inflate the cuff. Do not insert the tube too far into the trachea: the carina is not far from here.

- Ventilate the lungs with a standard self-inflating bag attached to high-flow oxygen. Exhalation occurs directly through the tracheal tube and tracheal suction is also now possible.

- Confirm correct tube placement by auscultation and capnography.

- As there is a high risk of secondary damage this method should only be used in patients deteriorating because of unmanageable airway problems.

KEY LEARNING POINTS

- When undertaken by someone with appropriate skills and experience, tracheal intubation is an effective airway management technique during cardiopulmonary resuscitation.

- In unskilled hands, prolonged interruptions of chest compressions, and the high risk of failure and other complications (e.g. unrecognised oesophageal intubation) make tracheal intubation attempts potentially harmful.

Section 4.
Basic mechanical ventilation

LEARNING OUTCOMES

To understand:

- the role of automatic ventilators in the peri-arrest period

There are very few studies that address specific aspects of ventilation during advanced life support. There are some data indicating that the ventilation rates delivered by healthcare personnel during cardiac arrest are excessive. Various small portable automatic ventilators may be used during resuscitation. They are usually gas powered. If an oxygen cylinder is used, both to supply the patient with oxygen and to power the ventilator, the contents may be used up rapidly. Most automatic resuscitators provide a constant flow of gas to the patient during inspiration; the volume delivered is dependent on the inspiratory time (a longer time provides a greater tidal volume). Because pressure in the airway rises during inspiration, these devices are often pressure-limited to protect the lungs against barotrauma. Expiration occurs passively into the atmosphere.

Set an automatic resuscitator initially to deliver a tidal volume of 6 ml kg^{-1} ideal body weight at 10 breaths min^{-1}. Some ventilators have co-ordinated markings on the controls to facilitate easy and rapid adjustment for patients of different sizes, and others are capable of sophisticated variation in respiratory pattern. In the presence of a spontaneous circulation, the correct setting will be determined by checking the patient's arterial blood gas values. If a tracheal tube or supraglottic airway has not been inserted, do not attempt chest compressions during the inspiratory phase. Once a tracheal tube has been inserted it is unnecessary to interrupt chest compressions during inspiration. If a supraglottic airway is inserted it may be necessary to synchronise chest compressions with the ventilator if an excessive leak is occurring.

KEY LEARNING POINTS

- Automatic ventilators may be a useful adjunct during cardiopulmonary resuscitation, although there are limited data on their use. Their safe use requires appropriate training.

FURTHER READING

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Chapter 8.

Cardiac monitoring, electrocardiography, and rhythm recognition

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LEARNING OUTCOMES

To understand:

- the reasons for ECG monitoring
- how to monitor the ECG
- the origin of the ECG
- the importance of recording the ECG
- the cardiac rhythms associated with cardiac arrest
- how to identify other common arrhythmias

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1.   Introduction

During cardiac arrest, identification of the cardiac rhythm will help to determine the correct treatment. Establish cardiac monitoring as soon as possible during cardiac arrest. In many patients who have been resuscitated from cardiac arrest there is a substantial risk of further arrhythmia and re-arrest. Maintain cardiac monitoring in people who have been resuscitated from cardiac arrest until you are confident that the risk of recurrence is very low.

Some patients present with an arrhythmia that may lead to cardiac arrest or other serious deterioration in their condition. Early detection and treatment of the arrhythmia may prevent cardiac arrest in some patients and prevent life-threatening deterioration in others. Patients at risk include those with persistent arrhythmia associated with structural heart disease, chest pain, heart failure, reduced conscious level or shock. In all patients with persistent cardiac arrhythmia at risk of deterioration, establish cardiac monitoring and whenever possible record a good-quality 12-lead ECG. Monitoring alone will not always allow accurate rhythm recognition and it is important to document the arrhythmia for future reference if required.

Some people experience symptoms (usually syncope) caused by an intermittent cardiac arrhythmia that, if not documented and treated, could lead to cardiac arrest or sudden death. However, the arrhythmia may not be present at the time of initial assessment. In people who present with syncope undertake careful clinical assessment and record a 12-lead ECG. People who have experienced uncomplicated faints, situational syncope (such as cough syncope or micturition syncope) or syncope due to orthostatic hypotension do not require cardiac monitoring and do not usually require hospital admission. In those who have had unexplained syncope, especially during exercise, those who have had syncope and have evidence of structural heart disease, and those who have had syncope and have an abnormal ECG (especially a prolonged QT interval or broad QRS ≥ 0.12 sec) start cardiac monitoring and arrange further expert cardiovascular assessment.

Single-lead ECG monitoring is not a reliable technique for detecting presence of myocardial ischaemia (ST segment depression/elevation or T waves changes). Record serial 12-lead ECGs in people experiencing chest pain suggestive of an acute coronary syndrome.

During cardiac arrest, recognition of ventricular fibrillation/pulseless ventricular tachycardia (VF/pVT) as shockable rhythms is crucial to the delivery of effective treatment. Automated external defibrillators (AEDs) and shock advisory defibrillators (SADs) can recognise these rhythms reliably by electronic analysis. If a shockable rhythm is present, the defibrillator will charge to the appropriate energy level and instruct the operator that a shock is required. The introduction of AEDs has enabled resuscitation from VF/pVT to be achieved by people who do not have skill in rhythm recognition, both in hospitals and in the community.

The accurate analysis of some cardiac rhythm abnormalities requires experience and expertise; however, the non-expert can interpret most rhythms sufficiently to select the appropriate treatment. The main priority is to recognise that the rhythm is abnormal and that the heart rate is inappropriately slow or fast. Use the structured approach to rhythm interpretation, described in this chapter, to avoid errors. The need for immediate treatment will be determined largely by the effect of the arrhythmia on the patient rather than by the nature of the arrhythmia. When an arrhythmia is present, first assess the patient (use the ABCDE approach), and then interpret the rhythm as accurately as possible. Treat the patient, not the ECG!

2. Techniques for ECG monitoring

2.1. Cardiac monitors

Cardiac monitors display the ECG on a screen in real time. The signal is obtained from adhesive electrodes on the patient's skin and transmitted to the monitor either by wires or by telemetry. Many monitor systems have other features, such as the ability to print samples of the ECG rhythm display or to store samples of the ECG. Most monitors include a display of heart rate, and some have alarms that can be programmed to provide an alert when the heart rate goes below or exceeds preset limits.

Many systems enable monitoring of other values such as blood pressure and oxygen saturation, which are important in the assessment of patients at risk. Digital processing of the ECG offers the potential for electronic analysis of the cardiac rhythm. If a patient requires monitoring, make sure that the monitor is being observed so that immediate action can be taken if needed.

2.1.1. How to attach the monitor

Attach ECG electrodes to the patient using the positions shown in figure 8.1. These will enable monitoring using 'modified limb leads' I, II and III. Make sure that the skin is dry, not greasy (use an alcohol swab and/or abrasive pad to clean), and either place the electrodes on relatively hair-free skin or shave off dense hair. Place electrodes over bone rather than muscle, to minimise interference from muscle artefact in the ECG signal. Different electrode positions may be used when necessary (e.g. trauma, recent surgery, skin disease).

Figure 8.1
Position of electrodes for monitoring the ECG using modified limb leads
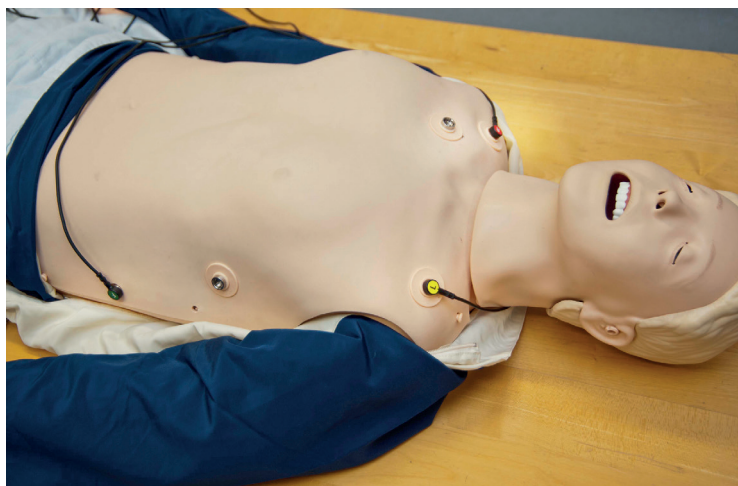
Most leads are colour-coded to help with correct connection. The usual scheme (except in the United States) uses **R**ed for the **R**ight arm lead, ye**LL**ow for the **L**eft arm lead, **G**reen for the le**G** lead (usually placed on the abdomen or lower left chest wall) for modified limb leads.

Sometimes a fourth Black electrode is available (usually placed on the right side of the abdomen or lower right chest wall).

Begin by monitoring in modified lead II as this usually displays good amplitude sinus P waves and good amplitude QRS complexes, but switch to another lead if necessary to obtain the best ECG signal. Try to minimise muscle and movement artefact by explaining to patients what the monitoring is for and by keeping them warm and relaxed.

2.1.2. Emergency monitoring

In an emergency, such as a collapsed patient, assess the cardiac rhythm as soon as possible by applying adhesive defibrillator pads, which can be used for monitoring and hands-free shock delivery *(figure 8.2)*. Apply the pads in the conventional positions, below the right clavicle and to the left axilla, in mid-axillary line. Use anterior and posterior positions as an alternative if the conventional positions cannot be used (e.g. permanent pacemaker in right pectoral position, chest wall trauma). The rapid application of manual defibrillator paddles also enables the cardiac rhythm to be determined rapidly, but in most healthcare environments these paddles have been replaced with hands-free adhesive defibrillator pads.

Figure 8.2
Defibrillator pads

3. Diagnosis from cardiac monitors

Use the displays and printouts from cardiac monitors only for rhythm recognition; do not attempt to interpret ST segment abnormalities or other more sophisticated elements of the ECG from monitors. When an arrhythmia is detected on a monitor, record a rhythm strip whenever possible.

If the arrhythmia persists for long enough, record a 12-lead ECG. It is not always possible to identify an arrhythmia from a single lead ECG recording. The heart is a three-dimensional organ and the 12-lead ECG examines the electrical signals from the heart in three dimensions. Sometimes, features that enable precise identification of cardiac rhythm are visible in only one or two leads of the 12-lead ECG and would not be seen on a single-lead recording of any other lead *(figure 8.3)*.

These recordings may assist with rhythm interpretation at the time but are also useful for later examination and planning of treatment in the longer term. Therefore effective management of any arrhythmia, including a cardiac arrest arrhythmia, includes good quality ECG recording, as well as interpretation and treatment at the time.

Valuable information about the nature and origin of a tachyarrhythmia can also be obtained by recording the response to treatment (e.g. carotid sinus massage, adenosine). Whenever possible, the effect of any such intervention should be recorded on a continuous ECG recording, if possible using multiple leads *(figure 8.4)*.

Figure 8.3
12-lead ECG showing atrial tachycardia, which may be recognised in leads V1,V2.

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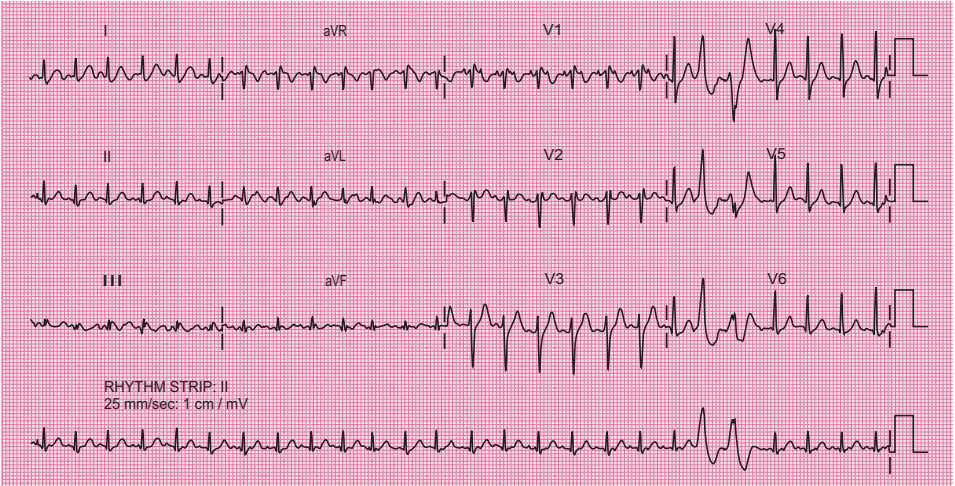
Figure 8.4
12-lead ECG showing the effect of adenosine in atrial flutter. Transient AV-block demonstrates clearly that this regular narrow-complex tachycardia was atrial flutter with 2:1 AV conduction.
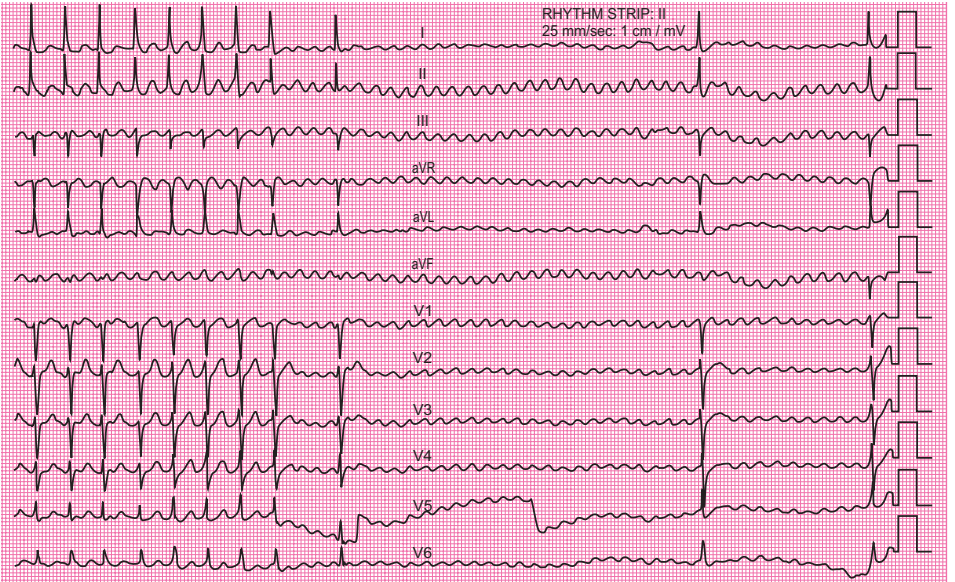
4. Basic electrocardiography

At rest, the cells of the cardiac conducting system and myocardium are polarised. A potential difference of approximately 90 mV is present between the inside of the cell (which is negatively charged) and the extracellular space. A sudden shift of ions across the cell membrane triggers depolarisation, generating the electrical signal that travels through the conducting system and triggers contraction of myocardial cells.

In normal sinus rhythm, depolarisation starts in a group of specialised 'pacemaker' cells, called the sino-atrial (SA) node, located close to the entry of the superior vena cava into the right atrium. A wave of depolarisation then spreads from the SA node through the atrial myocardium.

This is seen on the ECG as the P wave *(figure 8.5)*. Atrial contraction is the mechanical response to this electrical impulse.

The transmission of this electrical impulse to the ventricles occurs through specialised conducting tissue *(figure 8.6)*.

Figure 8.5
Components of the normal ECG signal
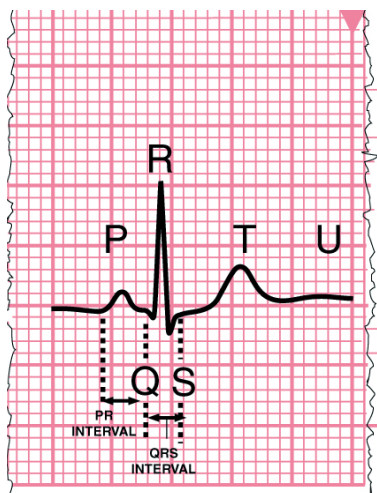
Figure 8.6
Electrical conduction in the heart
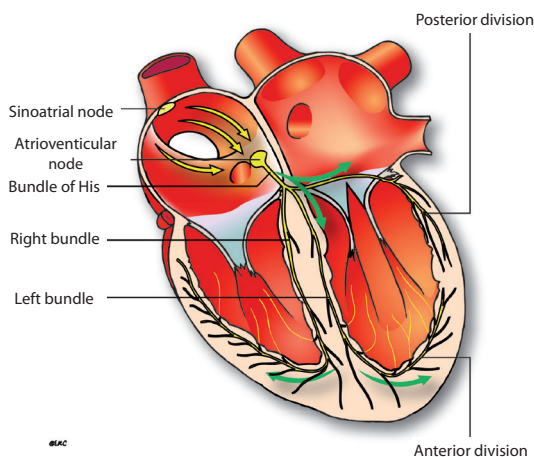
Firstly, there is slow conduction through the atrioventricular (AV) node, followed by rapid conduction to the ventricular myocardium by specialised conducting tissue (Purkinje fibres). The bundle of His carries these fibres from the AV node and then divides into right and left bundle branches, spreading out through the right and left ventricles respectively. Rapid conduction down these fibres ensures that the ventricles contract in a coordinated fashion.

Depolarisation of the bundle of His, bundle branches and ventricular myocardium is seen on the ECG as the QRS complex *(figure 8.5)*. Ventricular contraction is the mechanical response to this electrical impulse.

Between the P wave and QRS complex is a small isoelectric segment, which largely represents the delay in transmission through the AV node. The normal sequence of atrial depolarisation followed by ventricular depolarisation (P wave followed by QRS complex) is sinus rhythm *(rhythm strip 1)*.

The T wave, which follows the QRS complex, represents recovery of the resting potential in the cells of the conducting system and ventricular myocardium (ventricular repolarisation).

Because the normal conducting system transmits the depolarising impulse rapidly to both ventricles, the normal QRS complex is of relatively short duration (normally < 0.12 s).

When one of the bundle branches is diseased or damaged, rapid conduction to the corresponding ventricle is prevented. The depolarising impulse travels more rapidly down the other bundle branch to its ventricle and then more slowly, through ordinary ventricular myocardium to the other ventricle. This situation is called bundle branch block. Because depolarisation of both ventricles takes longer than normal it is seen on the ECG as a broad QRS complex (0.12 s or longer).

5.   How to read a rhythm strip

Experience and expertise may be needed to identify some rhythm abnormalities with complete precision. However, a simple, structured approach to interpreting the rhythm on any ECG recording will define any rhythm in sufficient detail to enable the most appropriate treatment to be chosen.

Apply the following 6-step approach to the analysis of any rhythm on an ECG:

1. Is there any electrical activity?

2. What is the ventricular (QRS) rate?

3. Is the QRS complex width normal or prolonged?

4. Is the QRS rhythm regular or irregular?

5. Is atrial activity present?

6. Is atrial activity related to ventricular activity and, if so, how?

Any cardiac rhythm can be described accurately (e.g. irregular narrow complex tachycardia, regular broad-complex bradycardia, etc.) and managed safely and effectively using the first four steps.

5.1.   Is there any electrical activity?

If you cannot see any electrical activity, check that the gain control is not too low and that the electrodes and leads are connected to both the patient and the monitor.

Check the patient: is a pulse present? If the patient is pulseless and there is still no activity on the ECG this is asystole *(rhythm strip 2)*. Atrial and ventricular asystole are often both present, resulting in a line with no deflections. A completely straight line indicates usually that a monitoring lead has become disconnected. Disconnection may also be displayed by a straight but discontinuous line. During asystole the ECG usually shows slight undulation of the baseline, and may show electrical interference due to respiratory movement, or chest compression.

Atrial activity (usually P waves but occasionally atrial fibrillation (AF) or atrial flutter) may continue for a short time after the onset of ventricular asystole. The ECG will show the atrial activity but no QRS complexes - ventricular standstill *(rhythm strip 3)*. Recognition of this is important because pacing is more likely to achieve a cardiac output in this situation than in most cases of complete asystole *(chapter 10)*.

If the patient is pulseless and electrical activity is present, decide whether recognisable QRS complexes are present. If not, and the ECG shows rapid, bizarre, irregular deflections of random frequency and amplitude, this is VF *(rhythm strip 4)*. In VF all coordination of electrical activity is lost, and there is no effective ventricular contraction, and no detectable cardiac output.

Ventricular fibrillation is sometimes classified as coarse *(rhythm strip 4)* or fine *(rhythm strip 5)* depending on the amplitude of the complexes; If there is doubt about whether the rhythm is asystole or fine VF, do not attempt defibrillation; instead, continue chest compressions and ventilation. Fine VF that is difficult to distinguish from asystole is unlikely to be shocked successfully into a rhythm that produces a cardiac output. Continuing good-quality CPR may improve the amplitude and frequency of the VF and improve the chance of subsequent successful defibrillation and return of spontaneous circulation. Delivering repeated shocks in an attempt to defibrillate what is thought to be fine VF will increase myocardial injury both directly from the electric current and indirectly from the interruptions in coronary blood flow *(chapter 6)*.

If electrical activity is present and contains recognisable QRS complexes, continue with the following steps in rhythm analysis.

If the patient is pulseless and there are recognisable complexes on the ECG that would be expected to produce a pulse, this is pulseless electrical activity (PEA) and requires immediate CPR. Do not delay CPR whilst the cardiac rhythm is analysed further.

5.2. What is the ventricular (QRS) rate?

The normal heart rate (ventricular rate) at rest is 60-100 beats min[-1]. A bradycardia has a heart rate slower than 60 min[-1]. A tachycardia has a rate faster than 100 min[-1]. ECG paper is calibrated in mm, with bolder lines every 5 mm. Standard paper speed is 25 mm s[-1]. One second is represented by 5 large squares (25 small squares). Be aware that in some countries standard paper speed is 50 mm sec[-1].

The best way of estimating the heart rate is to count the number of QRS complexes that occur in 6 s (30 large squares) and multiply by 10. This provides an estimate of heart rate, even when the rhythm is somewhat irregular. For example, if 20 QRS complexes occur in 30 large squares the rate is 200 min[-1] *(figure 8.7)*. For shorter rhythm strips count the number of QRS complexes in 3 s (15 large squares) and multiply by 20.

Figure 8.7
Calculation of heart rate from a rhythm strip (20 cardiac cycles occur in 30 large squares = 200 min[-1])

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5.3. Is the QRS complex width normal or prolonged?

The upper limit of normal for the QRS interval is 0.12 s (3 small squares). If the QRS width is less than this, the rhythm originates from above the bifurcation of the bundle of His and may be from the SA node, atria or AV node, but not from the ventricular myocardium. If the QRS duration is 0.12 s or more the rhythm may be coming from ventricular myocardium or may be a supraventricular rhythm, transmitted with aberrant conduction (i.e. bundle branch block).

5.4. Is the QRS rhythm regular or irregular?

This is not always as easy as it seems; at faster heart rates beat-to-beat variation during some irregular rhythms appears less obvious. Some rhythms may be regular in places but intermittent variation in R-R interval makes them irregular. Inspect an adequate length of rhythm strip carefully, measuring out each R-R interval and comparing it to others to detect any irregularity that is not obvious at first glance. Dividers are very useful for comparing the R-R intervals. Alternatively, the position of two adjacent identical points in the cardiac cycle (such as the tips of the R waves) can be marked on a strip of paper; this can then be moved to another section of the rhythm strip. If the rhythm is regular the marks will align precisely with each pair of R waves.

If the QRS rhythm is irregular, decide:

- Is this totally irregular, with no recognisable pattern of R-R interval?

- Is the basic rhythm regular, with intermittent irregularity?

- Is there a recurring cyclical variation in the R-R intervals?

If there is a cyclical pattern, the relationship between the QRS waves and the P wave requires careful analysis, as described below. If the R-R intervals are totally irregular (irregularly irregular) and the QRS complex is of constant morphology, the rhythm is most likely to be AF *(rhythm strip 6)*.

A regular underlying rhythm may be made irregular by extrasystoles (ectopic beats). Extrasystoles can arise from the atria or the ventricles, and the position or focus from which they arise will determine their morphology on an ECG.

If the QRS complex of ectopic beats is narrow (< 0.12 s), the beat is likely to have come from above the ventricular myocardium (i.e. from atrial muscle or the AV node).

Broad-complex ectopic beats may be of ventricular origin or may be supraventricular ectopic beats with bundle branch block.

Broad-complex atrial premature beats can sometimes be identified by a preceding ectopic P wave. Ventricular ectopic beats can be accompanied by a P wave occurring shortly after the QRS complex, caused by retrograde conduction from the ventricles to the atria.

Ectopic beats that occur early (that is before the next regular sinus beat was due to occur) are referred to as premature beats *(rhythm strip 7)*.

A beat that arises from the AV node or from ventricular myocardium after a long pause, for example during sinus bradycardia or after sinus arrest, is referred to as an escape beat *(rhythm strip 8)*. This implies that the focus in the AV node or ventricle that generates this beat is acting as a back-up pacemaker, because the normal pacemaker function of the sinus node is too slow or absent. Ectopic beats may occur singly, in pairs (couplets) or in threes (triplets). If more than three ectopic beats occur in rapid succession, this is regarded as a tachyarrhythmia.

An arrhythmia that occurs intermittently, interspersed with periods of normal sinus rhythm, is described as paroxysmal.

When ectopic beats occur alternately with sinus beats for a sustained period this is called bigeminy. It may be referred to as atrial bigeminy or ventricular bigeminy, depending on whether the ectopic beats are atrial or ventricular in origin.

5.5. Is atrial activity present?

Having defined the rhythm in terms of rate, regularity and QRS width, examine the ECG carefully for evidence of atrial activity. This may be difficult or impossible to identify, either because it is not visible or because atrial activity is partly or completely obscured by QRS

complexes or T waves. Do not guess or try to convince yourself that you can identify atrial activity unless you are completely sure.

Depending on the nature of the arrhythmia and the ECG lead being examined, P waves may be present as positive deflections, negative deflections or biphasic deflections. When present, U waves may be mistaken for P waves. P waves may coincide with and cause distortion or variation of QRS complexes, ST segments, or T waves. Whenever possible, recording of a 12-lead ECG may enable P waves to be identified in one or more leads, even if they cannot be seen clearly in the initial monitoring lead. Lead V1 is often useful for clear demonstration of some types of atrial activity including sinus P waves and AF. Sinus P waves are usually seen clearly in lead II.

Other types of atrial activity may be present. During atrial flutter, atrial activity is seen as flutter waves - an absolutely regular repetitive deflection with a 'saw-tooth' appearance, often at a rate of about 300 min^{-1}. This is usually seen best in the inferior leads (II, III, aVF) *(figure 8.4)*.

During AF, circuits and waves of depolarisation travel randomly through both atria. There are no P waves. Atrial fibrillation waves may be seen as rapid deviations from the baseline of varying amplitude and duration, usually seen best in lead V1. In some patients this may be of such low amplitude that no atrial activity can be seen.

During a sustained tachycardia atrial activity may not be visible between the QRS complexes. If the rhythm is of atrial origin (e.g. atrial flutter or AF) it may be possible to reveal atrial activity by slowing the ventricular rate whilst recording an ECG, preferably in multiple leads. For example, if a regular tachycardia of 150 min^{-1} is due to atrial flutter with 2:1 conduction it may not be possible to identify flutter waves with confidence. A transient increase in AV-block by vagal stimulation or by an intravenous bolus of adenosine will demonstrate the flutter waves and identify the rhythm accurately *(figure 8.4)*.

The shape and direction of P waves help to identify the atrial rhythm. For example, sinus P waves are upright in leads II and aVF. If retrograde activation of the atria is taking place from the region of the AV node (i.e. the rhythm is junctional or ventricular in origin), the P waves will be inverted in leads II and aVF because atrial depolarisation travels in the opposite direction to normal.

P wave rate and regularity (and flutter wave rate) are assessed in the same way as the rate and regularity of QRS complexes.

5.6.   Is atrial activity related to ventricular activity and, if so, how?

If there is a consistent interval between each P wave and the following QRS complex, it is likely that conduction between atrium and ventricle is intact and that ventricular depolarisation is triggered by atrial depolarisation. Examine a long rhythm strip to make sure that subtle variation in the PR interval is not missed.

Please note that the term 'PR interval' is used throughout the text, however it is recognised that some European countries use the PQ interval instead. In terms of rhythm interpretation, the two terms are interchangeable.

Occasionally conduction between atria and ventricles is reversed (i.e. ventricular depolarisation is followed by retrograde conduction through the AV node and then by atrial depolarisation); the P wave occurs soon after the QRS complex. It may sometimes be difficult to distinguish between this situation and the presence of a very long PR or PQ interval.

In other circumstances careful inspection will detect no relationship between the timing of P waves and of QRS complexes. This will indicate that atrial and ventricular depolarisation is arising independently, sometimes referred to as atrioventricular dissociation. Examples of this include:

- Complete (third degree) AV-block, where a normal sinus rate in the atria is accompanied by a regular bradycardia arising below the AV node.

- Some examples of VT in which regular broad QRS complexes are present and regular P waves can be seen at a different, slower rate, out of phase with the QRS complexes.

Difficulty may arise when the relationship between the P waves and the QRS complexes varies in a recurring pattern. This may be misinterpreted as atrioventricular dissociation. This is seen most commonly in one form of second degree AV-block (called Wenkebach or Mobitz I AV-block). Examine a long rhythm strip carefully for recurring patterns and plot and compare the timing of P waves and QRS complexes. In complete AV-block, the QRS rhythm is usually completely regular.

In AF, the atrial activity is completely irregular, so there is no identifiable relationship between this atrial activity and the irregular ventricular rhythm that results from it. If AF is accompanied by a completely regular, slow ventricular rhythm this is likely to be due to complete AV-block in the presence of AF in the atria.

In atrial flutter there may be a consistent relationship between the flutter waves and the QRS complexes, giving rise to 1:1, 2:1, 3:1 conduction etc. In some instances, there is a constantly varying relationship, producing an irregular QRS rhythm; this is atrial flutter with variable AV-block.

6. Cardiac arrest rhythms

The rhythms present during cardiac arrest can be classified into 3 groups:

- ventricular fibrillation (VF) and some cases of ventricular tachycardia (VT)

- asystole

- pulseless electrical activity (PEA)

Extreme bradycardia and rarely very fast supraventricular tachyarrhythmia may also cause such a severe fall in cardiac output to effectively cause cardiac arrest.

6.1. Ventricular fibrillation

The characteristic appearance of VF *(rhythm strip 4)* is usually easy to recognise, and this is the only rhythm that does not need the systematic rhythm analysis described earlier in this chapter. When a monitor appears to show VF check the patient immediately to establish whether this is VF requiring immediate defibrillation, or whether the appearance is due to artefact. If the patient has a pulse, the rhythm is not VF.

Two rhythm abnormalities may resemble VF in some circumstances, since both produce an irregular, broad-complex, fast rhythm:

> One is polymorphic VT *(rhythm strip 12)*. This may cause cardiac arrest, and when it does so the immediate treatment is the same as for VF, so failure to distinguish this immediately from VF would not lead to inappropriate treatment. However, it is important to document polymorphic VT and to recognise it following immediate resuscitation, so that the causes can be identified and corrected and appropriate treatment given to prevent recurrence.

> The second possible source of confusion is pre-excited AF. This occurs in the presence of an accessory pathway connecting atrial and ventricular muscle in the Wolff-Parkinson-White (WPW) syndrome. Some of these accessory pathways can conduct very rapidly, transmitting atrial impulses to the ventricles, sometimes at 300 min^{-1} or faster. This produces an irregular broad complex tachycardia with some variability in the width of QRS complexes *(figure 8.8)* that does not usually resemble VF but might be mistaken for polymorphic VT. Left untreated, this rhythm may lead to VT or VF causing cardiac arrest. If AF with WPW syndrome itself caused clinical cardiac arrest, the correct treatment would be immediate defibrillation (as for any broad-complex pulseless tachycardia) so misinterpretation as VT or VF would not lead to inappropriate treatment. Again, the importance of documenting and recognising the rhythm is to ensure that the patient receives immediate appropriate specialist referral for treatment to protect them against the risk of recurrence of this potentially dangerous arrhythmia.

6.2. Ventricular tachycardia

Ventricular tachycardia (VT) may cause loss of cardiac output resulting in cardiac arrest, particularly at faster rates or in the presence of structural heart disease (e.g. impaired left ventricular function, severe left ventricular hypertrophy, aortic stenosis). VT may degenerate suddenly into VF. Pulseless VT is treated in the same way as VF by immediate defibrillation.

In the presence of a cardiac output (i.e. palpable pulse), treatment of VT should follow the broad complex tachycardia algorithm described in chapter 11.

The QRS morphology may be monomorphic or polymorphic. In monomorphic VT *(rhythm strip 10)*, the rhythm is regular (or almost regular). The rate during VT may be anything from 100 to 300 min^{-1}, rarely faster. It is unusual to see more than slight variation in heart rate during any single episode of VT (other than in response to anti-arrhythmic drug therapy). Atrial activity may continue independently of ventricular activity; the identification of

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P waves, dissociated from QRS complexes during broad complex tachycardia, identifies the rhythm as VT. Occasionally these atrial beats may be conducted to the ventricles, causing capture beats or fusion beats *(rhythm strip 11)*. A capture beat produces a single normal-looking QRS complex during monomorphic VT, without otherwise interrupting the arrhythmia. In a fusion beat, a wave of depolarisation travelling down from the AV node occurs simultaneously with a wave of depolarisation travelling up from the ventricular focus producing the arrhythmia. This results in a hybrid QRS complex caused by fusion of the normal QRS complex with the complex of the monomorphic VT.

Figure 8.8
12-lead ECG showing pre-excited atrial fibrillation in a patient with Wolff-Parkinson-White syndrome
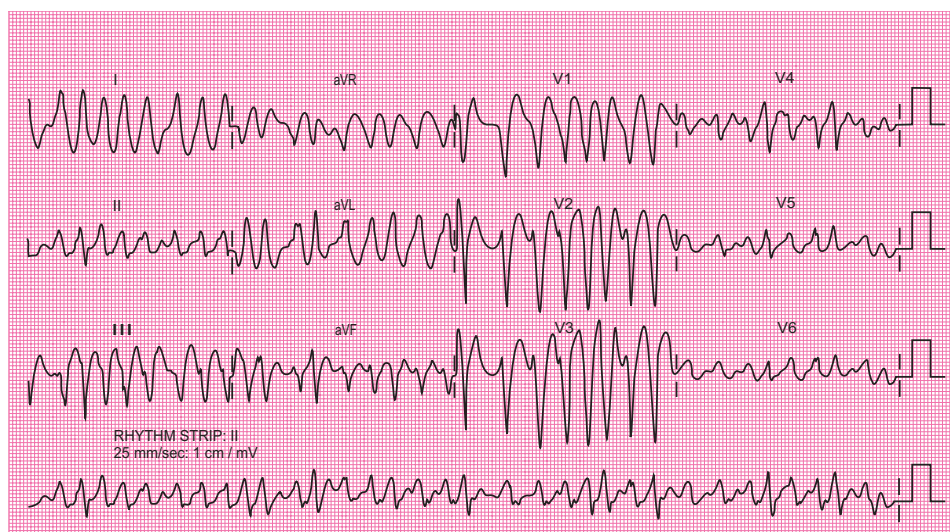
In the presence of bundle branch block, a supraventricular tachycardia (SVT) will produce a broad complex tachycardia. After myocardial infarction, most broad complex tachycardia will be ventricular in origin. The safest approach is to regard all broad complex tachycardia as VT until, or unless, proved otherwise.

One important type of polymorphic VT is torsade de pointes in which the axis of the electrical activity changes in a rotational way so that the overall appearance of the ECG on a rhythm strip produces a sinusoidal pattern *(rhythm strip 12)*. This arrhythmia usually arises in patients with a prolonged QT interval. This can occur as an inherited phenomenon in some families (long QT syndromes). In some people it is caused by drugs, including some anti-arrhythmic drugs, and it may occur less commonly as a manifestation of myocardial ischaemia. Many patients with TDP VT are also hypokalaemic and/or hypomagnesaemic. It is important to recognise TDP VT, because effective treatment (prevention of recurrent episodes) will require removal of any predisposing causes (i.e. drugs), treatment with intravenous magnesium and/or potassium, and may also require the use of overdrive

pacing. Drugs that prolong QT interval (including amiodarone) should be avoided in patients with TDP VT. This arrhythmia can itself cause cardiac arrest (in which case it is treated by defibrillation) and it can also degenerate into VF.

6.3. Asystole

The appearance of asystole has been described already *(rhythm strip 2)*. Sometimes it is not clear whether the observed rhythm is asystole or very fine VF. In this situation, immediate treatment is to provide high-quality CPR. If fine VF was present, good CPR may increase the amplitude and frequency of the VF, making that diagnosis clear and increasing the probability of successful defibrillation.

6.4. Pulseless electrical activity

The term pulseless electrical activity (PEA) does not refer to a specific cardiac rhythm. It defines the clinical absence of cardiac output despite electrical activity that would normally be expected to produce a cardiac output. It generally has a poor prognosis especially when it is caused by a very large acute myocardial infarction. Potentially more treatable causes include massive pulmonary embolism, tension pneumothorax, cardiac tamponade and acute severe blood loss.

7.   Peri-arrest arrhythmias

These are defined according to heart rate (bradyarrhythmia, tachyarrhythmia or arrhythmia with a normal rate), as this will dictate initial treatment *(chapter 11)*. In the unstable patient, concentrate on early treatment to prevent deterioration, rather than on prolonged attempts to identify the precise rhythm.

7.1. Bradyarrhythmia

A bradycardia is present when the ventricular (QRS) rate is < 60 min^{-1} *(rhythm strip 13)*. Bradycardia may be a physiological state in very fit people or during sleep, or may be an expected result of treatment (e.g. with a beta-blocker). Pathological bradycardia may be caused by malfunction of the SA node or from partial or complete failure of atrioventricular conduction. Some patients with these rhythm abnormalities may need treatment with an implanted pacemaker *(rhythm strip 14)*.

The emergency treatment of most bradycardia is with atropine and/or cardiac pacing. Occasionally it may be necessary to use sympathomimetic drugs such as isoprenaline or adrenaline. The need for treatment depends on the haemodynamic effect of the arrhythmia and the risk of developing asystole, rather than the precise ECG classification of the bradycardia. Extreme bradycardia may sometimes precede cardiac arrest and this may be prevented by prompt and appropriate treatment. In this context the most important bradyarrhythmia is acquired complete heart block *(see below)*.

7.1.1. Heart block: first degree atrioventricular block

The PR interval is the time between the onset of the P wave and the start of the QRS complex (whether this begins with a Q wave or R wave). The normal PR interval is between 0.12 and 0.20 s. First degree atrioventricular (AV) block is present when the PR interval is > 0.20 s and is a common finding *(rhythm strip 15)*. In some European countries, the PQ interval is used and for the purpose of this manual the term can be used interchangeably with 'PR interval'. It represents a delay in conduction through the AV junction (the AV node and bundle of His). In some instances this may be physiological (for example in trained athletes). There are many other causes of first degree AV-block, including primary disease (fibrosis) of the conducting system, various types of structural heart disease, ischaemic heart disease and use of drugs that delay conduction through the AV node. First degree AV-block rarely causes any symptoms and as an isolated finding rarely requires treatment.

7.1.2. Heart block: second degree atrioventricular block

Second degree AV-block is present when some, but not all, P waves are conducted to the ventricles, resulting in absence of a QRS complex after some P waves. There are two types:

- **Mobitz Type I AV-block (also called Wenckebach AV-block)**
The PR (or PQ) interval shows progressive prolongation after each successive P wave until a P wave occurs without a resulting QRS complex. Usually the cycle is then repeated *(rhythm strip 16)*.

Any condition that delays AV conduction can produce Wenkebach AV-block. In some situations this may be physiological, for example in highly trained athletes with high vagal tone. Outside that setting Wenckebach AV-block is usually pathological. Its many causes include acute myocardial infarction (especially inferior infarction). If asymptomatic, this rhythm does not usually require immediate treatment. The need for treatment is dictated by the effect of the bradyarrhythmia on the patient and the risk of developing more severe AV-block or asystole.

- **Mobitz Type II AV-block**
There is a constant PR (or PQ) interval in the conducted beats but some of the P waves are not followed by QRS complexes. This may occur randomly, without any consistent pattern. People with Mobitz II AV-block have an increased risk of progression to complete AV-block and asystole.

- **2:1 and 3:1 AV-block**
The term 2:1 AV-block describes the situation in which alternate P waves are followed by a QRS complex *(rhythm strip 17)*. 2:1 AV-block may be due to Mobitz I or Mobitz II AV-block and it may be difficult to distinguish which it is from the ECG appearance. If bundle branch block is present as well as 2:1 block (broad QRS complexes) this is likely to be Mobitz II block. 3:1 AV-block *(rhythm strip 18)* is less common and is a form of Mobitz II AV-block. Immediate decisions about treatment of these rhythms *(see algorithm for treatment of bradycardia - chapter 11)* will be determined by the effect of the resulting bradycardia on the patient. After identifying and providing any necessary immediate treatment continue cardiac monitoring and arrange expert cardiological assessment.

7.1.3. Heart block: third degree atrioventricular block

In third degree (complete) AV-block, there is no relationship between P waves and QRS complexes; atrial and ventricular depolarisation arises independently from separate 'pacemakers' *(rhythm strip 19)*. The site of the pacemaker stimulating the ventricles will determine the ventricular rate and QRS width. A pacemaker site in the AV node or proximal bundle of His may have an intrinsic rate of 40-50 min^{-1} or sometimes higher and may produce a narrow QRS complex. A pacemaker site in the distal His-Purkinje fibres or ventricular myocardium will produce broad QRS complexes, often have a rate of 30-40 min^{-1} or less, and is more likely to stop abruptly, resulting in asystole.

7.1.4. Escape rhythms

If the normal cardiac pacemaker (SA node) fails, or operates abnormally slowly, cardiac depolarisation may be initiated from a 'subsidiary' pacemaker in atrial myocardium, AV node, conducting fibres or ventricular myocardium. The resulting escape rhythm will be slower than the normal sinus rate. As indicated above, subsidiary pacemakers situated distally in the conducting system tend to produce slower heart rates than those situated more proximally. Thus a ventricular escape rhythm will usually be slower than a 'junctional' rhythm arising from the AV node or bundle of His.

The term idioventricular rhythm is used to describe a rhythm arising from ventricular myocardium. This includes ventricular escape rhythms seen in the presence of complete AV-block. The term accelerated idioventricular rhythm is used to describe an idioventricular rhythm with a normal heart rate (usually faster than the sinus rate but not fast enough to be VT). This type of rhythm is observed quite frequently after successful thrombolysis (or primary percutaneous coronary intervention) for acute myocardial infarction (a 'reperfusion arrhythmia'). Accelerated idioventricular rhythms do not influence prognosis unless they cause haemodynamic compromise or develop into VT or VF, which is uncommon. The QRS complex of an idioventricular rhythm will be broad (i.e. 0.12 s or greater), whereas a junctional rhythm may be narrow or broad, depending on whether conduction to the ventricles occurs normally, or with bundle branch block.

7.1.5. Agonal rhythm

Agonal rhythm occurs in dying patients. It is characterised by the presence of slow, irregular, wide ventricular complexes, often of varying morphology *(rhythm strip 20)*. This rhythm is seen commonly during the later stages of unsuccessful resuscitation attempts. The complexes slow inexorably and often become progressively broader before all recognisable activity is lost.

7.2. Tachyarrhythmia

A pathological tachycardia may arise from atrial myocardium, the AV junction or ventricular myocardium. Sinus tachycardia is not an arrhythmia and usually represents a response to some other physiological or pathological state (e.g. exercise, anxiety, blood loss, fever etc).

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7.2.1. Narrow-complex tachycardia

When a tachycardia arises from tissue situated above the bifurcation of the bundle of His, it is described as supraventricular *(rhythm strip 21)*. The QRS complexes will be narrow if ventricular depolarisation occurs normally, but will be broad if bundle branch block is present. QRS complexes may be regular in many rhythms or may be irregular in the presence of atrial fibrillation or variably conducted atrial flutter. Most tachycardia with narrow QRS complexes has a favourable prognosis, but the outlook will vary with individual clinical circumstances. These rhythms may be tolerated poorly by patients with structural heart disease and may provoke angina, especially in patients with coronary artery disease.

- **Atrial fibrillation**

Atrial fibrillation is the most common arrhythmia encountered in clinical practice. It is characterised by disorganised electrical activity in the atria. No recognisable P waves or co-ordinated atrial activity can be seen in any lead *(rhythm strip 6)*. The baseline is irregular and chaotic atrial activity is best seen in lead V1 where the atrial waveform is irregular in both amplitude and frequency. The QRS rhythm is irregularly irregular (i.e. there is no consistent R-R interval from beat to beat). The ventricular rate will depend on the refractory period of the AV junction. In the absence of drug treatment or pre-existing disease affecting the AV node, the resulting ventricular rate will be rapid, usually 120-180 min^{-1} or faster.

Common causes of AF include hypertension, obesity, alcohol excess and structural heart disease. In coronary heart disease AF usually results from left ventricular impairment (acute or chronic) and not as a direct result of ischaemia of the atrial myocardium.

- **Atrial flutter**

In atrial flutter, atrial activity is seen on the ECG as flutter or F waves at a rate of about 300 min^{-1} *(rhythm strip 22)*. These are best seen in the inferior leads II, III and aVF where they have a 'saw-tooth' appearance *(figure 8.4)*. The ventricular rate depends on AV conduction but there is often 2:1 *(rhythm strip 9)* or 3:1 conduction (often referred to as atrial flutter with 2:1 or 3:1 block). If conduction is constant the ventricular rhythm will be regular, but variable conduction causes an irregular ventricular rhythm. Like atrial fibrillation, atrial flutter is often, but not always, associated with underlying disease. Atrial flutter usually arises in the right atrium so is a recognised complication of diseases that affect the right heart, including chronic obstructive pulmonary disease, major pulmonary embolism, complex congenital heart disease and chronic congestive heart failure of any cause. It is also seen in patients after cardiac surgery.

7.2.2. Broad-complex tachycardia

Broad-complex tachycardia may be:

- a tachycardia arising in the ventricle below the bifurcation of the bundle of His, i.e. VT *(rhythm strip 10)*; or

- a supraventricular tachycardia conducted aberrantly (right or left bundle branch block) to the ventricles

The clinical consequences depend on:

- heart rate during the arrhythmia

- the presence or absence of structural heart disease or coronary disease

- duration of the arrhythmia

Ventricular tachycardia may degenerate into VF, especially if the VT is very fast (e.g. 200 min^{-1} or faster) or if the heart is unstable as a consequence of acute ischaemia or infarction, or in the presence of electrolyte abnormality (hypokalaemia or hypomagnesaemia).

Treat all broad-complex tachycardia as ventricular tachycardia unless there is good evidence that it is supraventricular in origin.

Patients with WPW syndrome have accessory pathways connecting atrial and ventricular myocardium. Some atrioventricular conduction occurs through these pathways as well as through the AV node. This results in widening of the QRS complexes by so-called delta waves. In the presence of such an accessory pathway that bypasses the AV node, AF may result in a ventricular rate that is so fast that cardiac output decreases dramatically. The ECG appearances are of a very rapid, irregular, broad complex tachycardia that usually shows variability in the width of QRS complexes. This rhythm may be misdiagnosed as irregular VT or possibly as VF. Overall the rhythm is more organised than ventricular fibrillation and lacks the random chaotic activity of variable amplitude.

8.   The QT interval

When identifying and treating rhythm abnormalities it is important to recognise likely underlying causes that may influence choice of effective treatment. These may be identified from clinical assessment (e.g. myocardial infarction), laboratory tests (e.g. electrolyte abnormality) or from the ECG. Prolongation of the QT interval predisposes people to ventricular arrhythmia, in particular VT and VF.

The QT interval is measured from the start of the QRS complex to the end of the T wave. It can be difficult to measure accurately, mainly because it may be difficult to identify the end of the T wave. This may be especially difficult when prominent U waves are present, merging with the end of the T wave. U waves can be a feature of some abnormalities (e.g. hypokalaemia) but may be present in some healthy people with normal hearts.

The length of the QT interval may also vary between different leads of the same ECG. This may partly reflect variation in amplitude and direction of the T wave, making it more difficult to measure in some leads than others. Variation in the QT interval (QT dispersion) has also been shown to be associated with an increased risk of death in patients with ischaemic heart disease, but this finding has not been developed into a useful measurement for use in clinical practice.

The QT interval varies with age, with gender and in particular with heart rate. The QT interval shortens as the heart rate increases. A correction can be made to allow for this,

using the measured QT interval and heart rate to calculate the corrected QT interval (QTc). The upper end of the normal range for QTc is 0.42 s. Many modern ECG machines measure the QT and other intervals and calculate the QTc automatically. These measurements are accurate only if the ECG recording is of good quality. Most ECG machines cannot distinguish between T waves and U waves. Always look at the recording and make sure that the quoted measurements are not obviously inaccurate. If in doubt seek expert help with interpretation.

Abnormality of the QT interval can be seen in various situations. A shortened QT interval may be seen with hypercalcaemia and digoxin treatment. Hypokalaemia, hypomagnesaemia, hypocalcaemia, hypothermia, myocarditis and in some instances myocardial ischaemia can all cause QT prolongation. There is also a long list of drugs that may prolong the QT interval, including class I and class III anti-arrhythmic drugs.

There are several genetic abnormalities in which the QT interval is abnormal or there is abnormality of ventricular repolarisation (principally the long QT, short QT and Brugada syndromes). The abnormality of repolarisation places them at risk of ventricular arrhythmia and sudden death. These people require expert assessment to identify whether treatment is needed to reduce this risk. For some the only effective treatment is an implantable cardioverter-defibrillator to treat VF or VT immediately, if it occurs. It is especially important that patients with long QT syndromes are not given any drug that may cause further QT prolongation.

KEY LEARNING POINTS

- A systematic approach to ECG rhythm analysis enables accurate recognition of any rhythm abnormality, sufficient to enable safe, effective treatment.

- Recordings of any rhythm abnormality and of the ECG in sinus rhythm provide valuable diagnostic information and help the correct choice of longer-term treatment.

- Accurate monitoring of the cardiac rhythm is essential for any patient at high risk of developing life-threatening arrhythmia.

- Accurate monitoring of the cardiac rhythm is essential in the management of cardiac arrest.

FURTHER READING

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- Fuster V, Ryden L E, Cannom DS, et al. ACC/AHA/ESC 2006 Guidelines for the Management of Patients with Atrial Fibrillation: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines (Writing Committee to Revise the 2001 Guidelines for the Management of Patients With Atrial Fibrillation): developed in collaboration with the European Heart Rhythm Association and the Heart Rhythm Society. Circulation 2006;114:e257-354. www.escardio.org
- Zipes DP, Camm AJ, Borggrefe M, et al. ACC/AHA/ESC 2006 guidelines for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: a report of the American College of Cardiology/American Heart Association Task Force and the European Society of Cardiology Committee for Practice Guidelines (Writing Committee to Develop Guidelines for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death) J Am Coll Cardiol 2006;48:e247-e346. www.escardio.org

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Rhythm Strip 1
Normal sinus rhythm

Rhythm Strip 2
Asystole
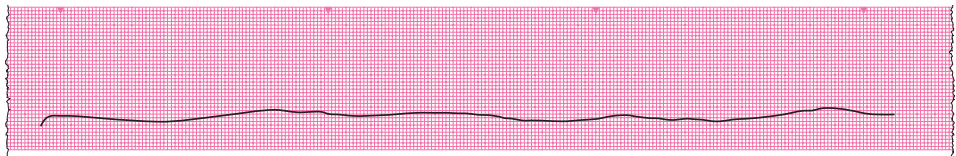
Rhythm Strip 3
P-wave asystole
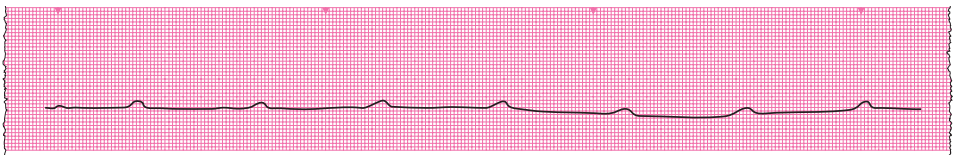
Rhythm Strip 4
Coarse ventricular fibrillation
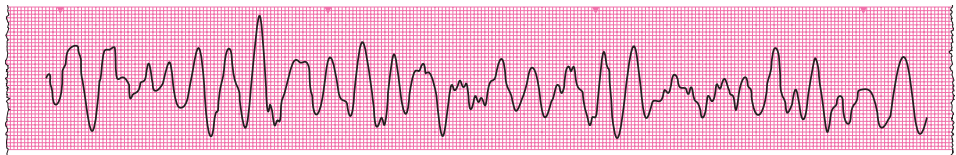
Rhythm Strip 5
Fine ventricular fibrillation
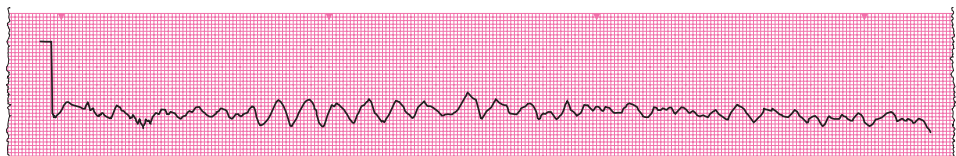
Rhythm Strip 6
Atrial fibrillation
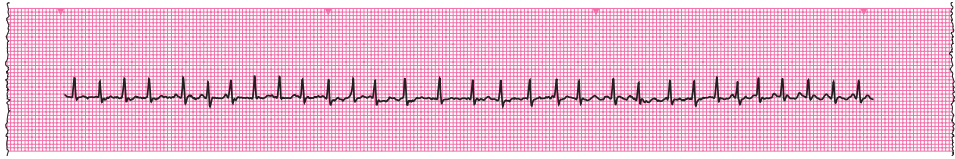
Rhythm Strip 7
Premature ventricular beat

8
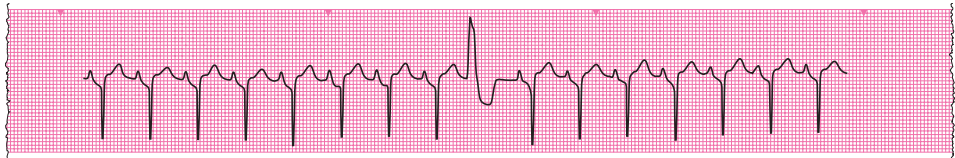
Rhythm Strip 8
Junctional escape beat

Rhythm Strip 9
Atrial flutter with 2:1 atrioventricular block

Rhythm Strip 10
Monomorphic ventricular tachycardia
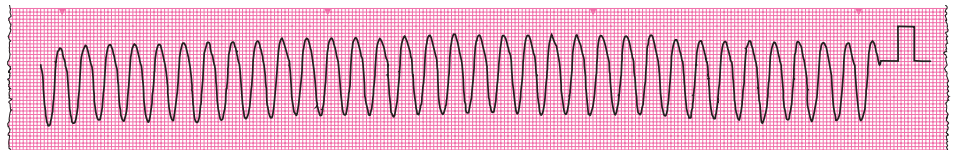
Rhythm Strip 11
Ventricular tachycardia with capture and fusion beats
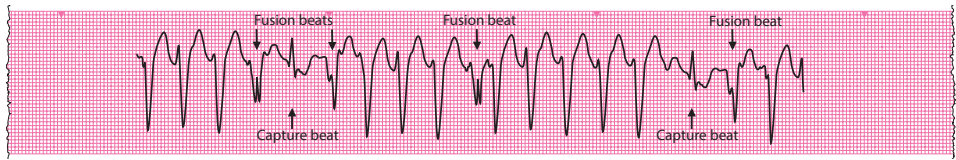
Rhythm Strip 12
Polymorhpic ventricular tachycardia - Torsade de Pointes (TdP)
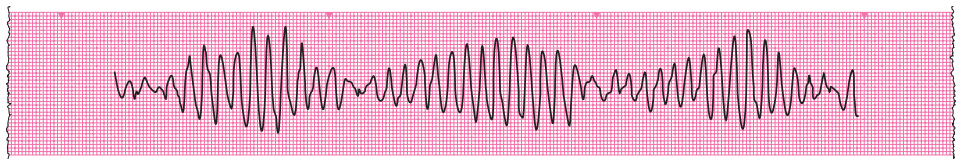
Rhythm Strip 13
Sinus bradycardia
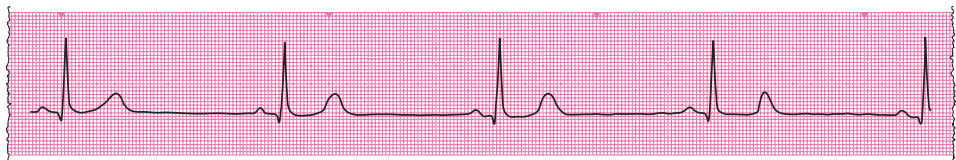
Rhythm Strip 14
Paced rhythm
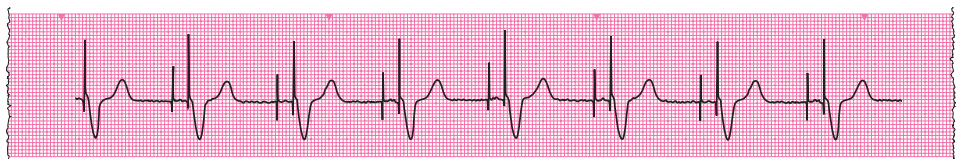
Rhythm Strip 15
First degree atrioventricular block

Rhythm Strip 16
Mobitz type I or Wenckebach block
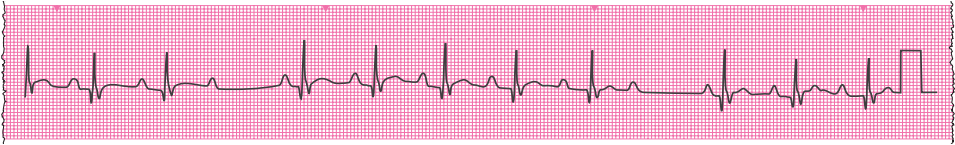
Rhythm Strip 17
Mobitz type II second degree atrioventricular block (2:1)

Rhythm Strip 18
Mobitz type II second degree atrioventricular block (3:1)

8
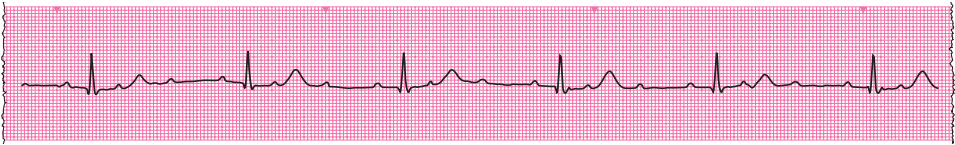
Rhythm Strip 19
Third degree (complete) atrioventricular block

Rhythm Strip 20
Agonal rhythm
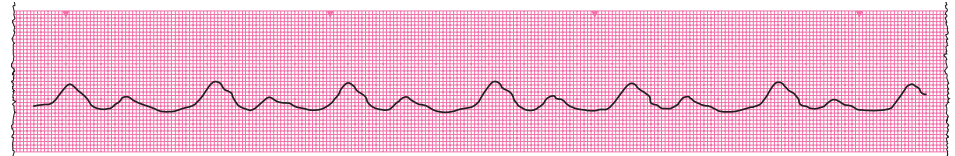
Rhythm Strip 21
Supraventricular tachycardia
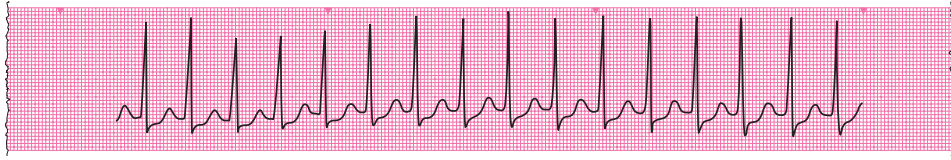
Rhythm Strip 22
Atrial flutter with a high degree of atrioventricular block
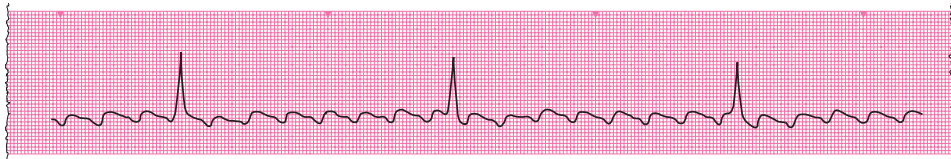
Chapter 9.

Defibrillation

<div>

LEARNING OUTCOMES

To understand:

- the mechanism of defibrillation
- the factors affecting defibrillation success
- the importance of minimising interruptions in chest compressions during defibrillation
- how to deliver a shock safely using either a manual or automated external defibrillator (AED)

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1. Introduction

Following the onset of ventricular fibrillation or pulseless ventricular tachycardia (VF/pVT), cardiac output ceases and cerebral hypoxic injury starts within 3 min. If complete neurological recovery is to be achieved, early successful defibrillation with a return of spontaneous circulation (ROSC) is essential. Defibrillation is a key link in the chain of survival and is one of the few interventions that has been shown to improve outcome from VF/pVT cardiac arrest. The probability of successful defibrillation declines rapidly with time; therefore early defibrillation is one of the most important factors in determining survival from cardiac arrest. In the absence of bystander CPR, for every minute that passes between collapse and attempted defibrillation, mortality increases 10-12 %. **The shorter the interval between the onset of VF/pVT and delivery of the shock, the greater the chance of successful defibrillation and survival.** Although defibrillation is key to the management of patients in VF/pVT, continuous, uninterrupted chest compressions are also required to optimise the chances of successful resuscitation. Analysis of CPR performance during out-of-hospital and in-hospital cardiac arrest has shown that significant interruptions are common and every effort should be made to minimise interruptions. The aim should be to ensure that chest compressions are performed continuously throughout the resuscitation attempt, pausing only to enable specific interventions.

Another factor that is critical in determining the success of defibrillation is the duration of the interval between stopping chest compressions and delivering the shock: the pre-

shock pause. The duration of the pre-shock pause is related inversely to the chance of successful defibrillation; every 5-second increase in the pre-shock pause almost halves the chance of successful defibrillation (defined by the absence of VF 5 s after shock delivery). Consequently, defibrillation must always be performed quickly and efficiently in order to maximise the chances of successful resuscitation.

If there is any delay in obtaining a defibrillator, and while the defibrillator is applied, start chest compressions and ventilation immediately. When bystander CPR is given, the decrease in survival is more gradual and averages 3-4% per minute from collapse to defibrillation. Bystander CPR can double survival from witnessed cardiac arrest.

2. Mechanism of defibrillation

Defibrillation is the passage of an electrical current of sufficient magnitude across the myocardium to depolarise a critical mass of cardiac muscle simultaneously, enabling the natural pacemaker tissue to resume control. To achieve this, all defibrillators have three features in common: a power source capable of providing direct current, a capacitor that can be charged to a pre-determined energy level and two electrodes which are placed on the patient's chest, either side of the heart, across which the capacitor is discharged. Successful defibrillation is defined scientifically as the absence of VF/pVT at 5 s after shock delivery, although the ultimate goal is ROSC.

3. Factors affecting defibrillation success

Defibrillation success depends on sufficient current being delivered to the myocardium. However, the delivered current is difficult to determine because it is influenced by transthoracic impedance (electrical resistance) and electrode position. Furthermore, much of the current is diverted along non-cardiac pathways in the thorax and, as a result, as little as 4% reaches the heart.

3.1. Transthoracic impedance

Current flow is inversely proportional to transthoracic impedance. Defibrillation technique must be optimised to minimise the transthoracic impedance in order to maximise delivery of current to the myocardium. In adults, impedance is normally in the range 70-80 ohm, but in the presence of poor technique may rise to 150 ohm, reducing the current delivered and thereby decreasing the chance of successful defibrillation. Transthoracic impedance is influenced by: electrode-to-skin contact, electrode size and phase of ventilation. Modern biphasic defibrillators can measure the transthoracic impedance and adjust the energy delivered to compensate and are therefore less susceptible to higher transthoracic impedance (impedance compensation).

The presence of a transdermal drug patch on the patient's chest may prevent good contact and may cause arcing and burns if self-adhesive pads are placed over them; if removing them and wiping the area dry before applying the electrodes is likely to delay defibrillation, place the pads in an alternative position that avoids the patch.

3.1.1. Shaving the chest

It may be difficult to obtain good electrode-to-skin contact in patients with very hairy chests. This increases impedance, reduces defibrillation efficacy and may cause burns to the patient's chest. If a patient has a very hairy chest, and if a razor is available immediately, use it to remove excessive hair from the area where the electrodes are placed. However, defibrillation should not be delayed if a razor is not at hand immediately. In very hairy patients, a bi-axillary electrode position may enable more rapid defibrillation.

3.1.2. Electrode size

The optimal electrode size is unknown. Current recommendations are that the sum of the electrode area should be a minimum of 150 cm^2. Self-adhesive pads 8-12 cm in diameter are widely used and function well. In practice the self-adhesive pads recommended by the manufacturer for the specific defibrillator should be used.

3.1.3. Respiratory phase

Transthoracic impedance varies during ventilation being minimal at end expiration. If possible, defibrillation should be attempted at this point. Positive end-expiratory pressure (PEEP) increases impedance and where possible should be minimised during defibrillation. Auto-PEEP (gas trapping) may be particularly high in asthmatics and may necessitate higher than usual energy levels for defibrillation.

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3.2. Electrode position

Transmyocardial current during defibrillation is likely to be maximal when the electrodes are placed so that the area of the heart that is fibrillating lies directly between them (i.e. ventricles in VF/pVT, atria in atrial fibrillation (AF)). Therefore, the optimal electrode position may not be the same for ventricular and atrial arrhythmias.

When attempting to defibrillate a patient in VF/pVT, the standard procedure is to place one electrode to the right of the upper sternum below the clavicle. The apical pad is placed in the mid-axillary line, approximately level with the V6 ECG electrode or female breast. This position should be clear of any breast tissue. It is important that this electrode is placed sufficiently laterally *(figure 9.1)*. Although the electrodes are marked positive and negative, each can be placed in either position. Other acceptable pad positions include:

- each electrode on the lateral chest walls, one on the right and the other on the left side (bi-axillary)

- one electrode in the standard apical position and the other on the right upper back

- one electrode anteriorly, over the left precordium, and the other electrode on the back behind the heart, just inferior to the left scapula (antero-posterior)

3.3. CPR or defibrillation first?

In any unwitnessed cardiac arrest, those responding should provide high-quality, uninterrupted CPR while a defibrillator is retrieved, applied and charged. Defibrillation must be performed as soon as possible, and a specific period of CPR (e.g. 2-3 min) before rhythm analysis and shock delivery is not recommended.

Figure 9.1
Standard electrode positions for defibrillation
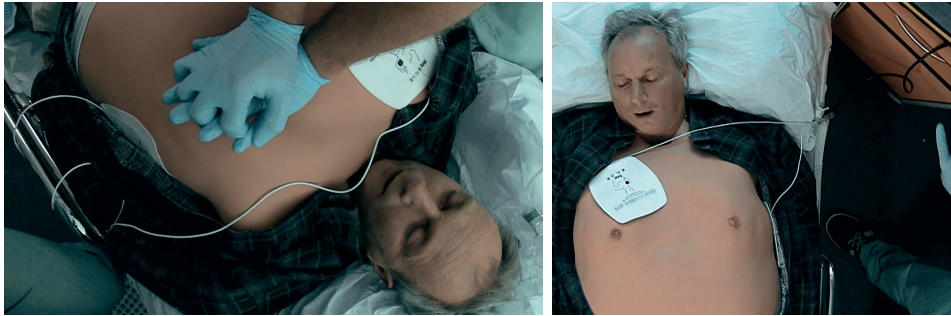
3.3.1. Strategies for minimising the pre-shock pause

The delay between stopping chest compressions and delivery of the shock (the pre-shock pause) must be kept to an absolute minimum; even 5-10 seconds delay will reduce the chances of the shock being successful. The pre-shock pause can be reduced to less than 5 seconds by continuing compressions during charging of the defibrillator and by having an efficient team coordinated by a leader who communicates effectively. The safety check to avoid rescuer contact with the patient at the moment of defibrillation should be undertaken rapidly but efficiently. The post shock pause is minimised by resuming chest compressions immediately after shock delivery. The entire process of manual defibrillation should be achievable with less than a 5 second interruption to chest compressions.

3.4. Shock sequence

With first-shock efficacy of biphasic waveforms generally exceeding 90 %, failure to cardiovert VF successfully suggests the need for a period of CPR to perfuse the myocardium, rather than a further shock. Thus, immediately after giving a single shock, and without reassessing the rhythm or feeling for a pulse, resume CPR (30 compressions : 2 ventilations) for 2 min before delivering another shock *(if indicated - see below)*. Even if the defibrillation attempt is successful in restoring a perfusing rhythm, it is very rare for a pulse to be palpable immediately after defibrillation and the delay in trying to palpate a pulse will further compromise the myocardium if a perfusing rhythm has not been restored. If a perfusing rhythm has been restored, giving chest compressions does not increase the chance of VF recurrence. In the presence of post-shock asystole, chest compressions may induce VF.

3.4.1. Witnessed, monitored VF/pVT in the cardiac catheter laboratory or after cardiac surgery

If a patient has a monitored and witnessed cardiac arrest in the catheter laboratory, coronary care unit, a critical care area or whilst monitored after cardiac surgery, and a manual defibrillator is rapidly available:

- confirm cardiac arrest and shout for help

- if the initial rhythm is VF/pVT, give up to three quick successive (stacked) shocks

- rapidly check for a rhythm change and if appropriate ROSC after each defibrillation attempt

- start chest compressions and continue CPR for two minutes if the third shock is unsuccessful

This three-shock strategy may also be considered for an initial, witnessed VF/pVT cardiac arrest if the patient is already connected to a manual defibrillator. Although there are no data supporting a three-shock strategy in any of these circumstances, it is unlikely that chest compressions will improve the already very high chance of ROSC when defibrillation occurs early in the electrical phase, immediately after onset of VF.

3.5. Shock energy and waveforms

Defibrillation requires the delivery of sufficient electrical energy to defibrillate a critical mass of myocardium, abolish the wavefronts of VF and enable restoration of spontaneous synchronised electrical activity in the form of an organised rhythm. The optimal energy for defibrillation is that which achieves defibrillation whilst causing the minimum of myocardial damage. Selection of an appropriate energy level also reduces the number of repetitive shocks, which in turn limits myocardial damage.

Optimal energy levels for defibrillation are unknown. The recommendations for energy levels are based on a consensus following careful review of the current literature. Although delivered energy levels are selected for defibrillation, it is the transmyocardial current flow that achieves defibrillation. Current correlates well with successful defibrillation and cardioversion.

There remains no evidence to support either a fixed or escalating energy protocol, although an escalating protocol may be associated with a lower incidence of refibrillation. Both strategies are acceptable; however, if the first shock is not successful and the defibrillator is capable of delivering shocks of higher energy it is reasonable to increase the energy for subsequent shocks.

3.5.1. Biphasic defibrillators

Biphasic waveforms, are now well established as a safe and effective waveform for defibrillation. Biphasic defibrillators compensate for the wide variations in transthoracic impedance by electronically adjusting the waveform magnitude and duration to ensure

optimal current delivery to the myocardium, irrespective of the patient's size (impedance compensation). There are two main types of biphasic waveform: the biphasic truncated exponential (BTE) and rectilinear biphasic (RLB).

Due to the higher first shock success rate for termination of fibrillation with a biphasic waveform and the potential for less post shock myocardial dysfunction biphasic waveforms should be used for cardioversion of both atrial and ventricular arrhythmias in preference to a monophasic waveform.

Figure 9.2

Biphasic truncated exponential waveform
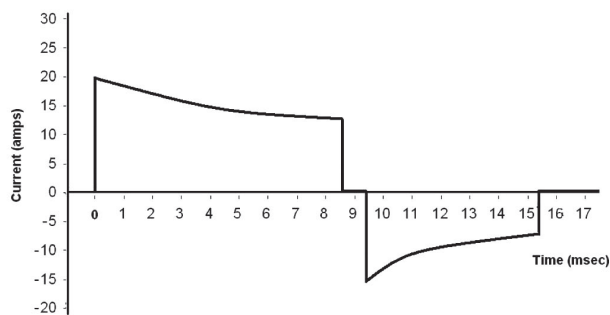
Figure 9.3

Rectilinear biphasic waveform
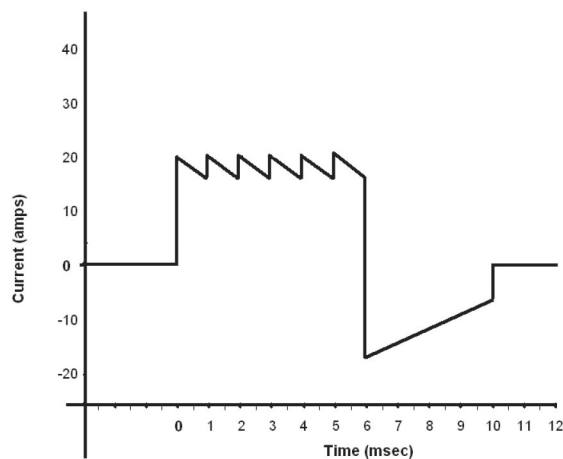
There is no evidence that either of the two most commonly used biphasic waveforms is more effective. Although the initial biphasic shock energy should be no lower than 120 J for a RLB waveform or 150 J for BTE waveforms, it is recommended that the initial biphasic shock should be at least 150 J for simplicity, irrespective of the biphasic waveform.

If the provider is unaware of the type of defibrillator (monophasic or biphasic) or its effective dose range, use the highest available energy for the first and subsequent shocks. If the first shock is unsuccessful, second and subsequent shocks can be delivered using either fixed or escalating energies of between 150-360 J, depending on the device in use. If a shockable rhythm recurs after successful defibrillation (with or without ROSC), give the next shock with the energy level that had previously been successful or higher.

3.5.2. Monophasic defibrillators

The monophasic waveform does not defibrillate as effectively as the biphasic waveform. Therefore, when using a monophasic defibrillator use 360 J for the first and all subsequent shocks.

3.6. Importance of uninterrupted chest compressions

The importance of early, uninterrupted chest compression is emphasised throughout this manual; they should be interrupted only for rhythm checking and shock delivery, and resumed as soon as a shock has been delivered. When two rescuers are present, the rescuer operating the defibrillator applies the electrodes whilst CPR is in progress. With manual defibrillation, it is possible to perform CPR during charging thereby reducing the pre-shock pause (interval from stopping compressions to shock delivery) to < 5 s. When using manual defibrillation, the entire process of pausing chest compressions, standing clear, delivering the shock and immediately resuming chest compressions should be achievable in < 5 s.

4. Safety

Attempted defibrillation should be undertaken without risk to members of the resuscitation team. This is achieved best by using self-adhesive pad electrodes as this eliminates the possibility of anyone touching any part of the electrode. Be wary of wet surroundings or clothing - wipe any water from the patient's chest before attempted defibrillation. No part of any person should make direct or indirect contact with the patient. Do not hold intravenous infusion equipment or the patient's trolley during shock delivery. The operator must ensure that everyone is clear of the patient before delivering a shock.

Gloves may provide limited protection from the electric current; therefore it is strongly recommended that all members of the resuscitation team wear gloves.

4.1. Safe use of oxygen during defibrillation

In an oxygen-enriched atmosphere, sparking from poorly applied defibrillator paddles can cause a fire and significant burns to a patient. The use of self-adhesive pads is far less likely to cause sparks than manual paddles - no fires have been reported in association with the use of self-adhesive pads. The following are recommended as good practice:

- Take off any oxygen mask or nasal cannulae and place them at least 1 m away from the patient's chest.

- Leave the ventilation bag connected to the tracheal tube or supraglottic airway, ensuring that there is no residual PEEP remaining in the circuit.

- If the patient is connected to a ventilator leave the ventilator tubing (breathing circuit) connected to the tracheal tube unless chest compressions prevent the ventilator from delivering adequate tidal volumes. During normal use, when connected to a tracheal tube, oxygen from a ventilator in the critical care unit will be vented from the main ventilator housing well away from the defibrillation zone. Patients in the critical care unit may be dependent on positive end expiratory pressure (PEEP) to maintain adequate oxygenation; during cardioversion, when the spontaneous circulation potentially enables blood to remain well oxygenated, it is particularly appropriate to leave the critically ill patient connected to the ventilator during shock delivery.

5. Automated external defibrillators

Automated external defibrillators are computerised devices that use voice and visual prompts to guide lay rescuers and healthcare professionals to attempt defibrillation safely in cardiac arrest victims. Advances in technology, particularly with respect to battery capacity, and software arrhythmia analysis have enabled the mass production of relatively cheap, reliable and easily operated portable defibrillators. Shock-advisory defibrillators have ECG-analysis capability but can usually be manually over-ridden by healthcare providers capable of rhythm recognition.

AEDs are safe and effective when used by laypeople with minimal or no training. AEDs make it possible to defibrillate many minutes before professional help arrives. CPR providers should continue CPR with minimal interruption of chest compressions while attaching an AED and during its use. CPR providers should concentrate on following the voice prompts immediately when they are spoken, in particular resuming CPR as soon as instructed, and minimizing interruptions in chest compression. Standard AEDs are suitable for use in children older than 8 years.

5.1. Automated rhythm analysis

Automated external defibrillators have microprocessors that analyse several features of the ECG, including frequency and amplitude. Some AEDs are programmed to detect spontaneous movement by the patient or others. Developing technology should soon enable AEDs to provide information about frequency and depth of chest compressions during CPR that may improve resuscitation performance by all rescuers.

Automated external defibrillators have been tested extensively against libraries of recorded cardiac rhythms and in many trials in adults and children. They are extremely accurate in rhythm analysis. Although AEDs are not designed to deliver synchronised shocks, all AEDs will recommend shocks for VT if the rate and R-wave morphology exceed preset values.

5.2.   In-hospital use of AEDs

Delayed defibrillation may occur when patients sustain cardiac arrest in unmonitored hospital beds and in outpatient departments. In these areas several minutes may elapse before resuscitation teams arrive with a defibrillator and deliver shocks.

Despite limited evidence, AEDs should be considered for the hospital setting as a way to facilitate defibrillation as soon as possible (within 3 min of collapse at the most) especially in areas where staff have no rhythm recognition skills or where they use defibrillators infrequently. An effective system for training and retraining should be in place. Adequate numbers of staff should be trained to enable achievement of the goal of providing the first shock within 3 min of collapse anywhere in the hospital.

Training in the use of AEDs can be achieved much more rapidly and easily than for manual defibrillators. Automated equipment has made attempted defibrillation available to a much wider range of medical, nursing, paramedical, and lay workers (e.g. police and first-aiders - 'first-responder defibrillation'). Healthcare providers with a duty to perform CPR should be trained, equipped, and authorised to perform defibrillation. First-responder attempted defibrillation is vital, as the delay to delivery of the first shock is the main determinant of survival in cardiac arrest.

5.3.   Public access defibrillation (PAD) programmes

Public access defibrillation (PAD) and first responder AED programmes may increase the number of victims who receive bystander CPR and early defibrillation, thus improving survival from out-of-hospital cardiac arrest. These programmes require an organised and practised response with rescuers trained and equipped to recognise emergencies, activate the EMS system, provide CPR, and use the AED. Lay rescuer AED programmes with very rapid response times in airports, on aircraft, or in casinos, and uncontrolled studies using police officers as first responders have achieved reported survival rates as high as 49-74 %. Recommended elements for PAD programmes include:

- a planned and practised response
- training of anticipated rescuers in CPR and use of the AED
- link with the local EMS system
- programme of continuous audit (quality improvement)

Public access defibrillation programmes are most likely to improve survival from cardiac arrest if they are established in locations where witnessed cardiac arrest is likely to occur. Suitable sites might include airports, casinos and sports facilities. Approximately 80 % of out-of-hospital cardiac arrests occur in private or residential settings; this fact inevitably limits the overall impact that PAD programmes can have on survival rates.

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Figure 9.4
AED algorithm

```
                    ┌─────────────────────────┐
                    │      Unresponsive?       │─────────►  ┌──────────────┐
                    └─────────────────────────┘            │ Call for help │
                                 │                          └──────────────┘
                                 ▼
                    ┌─────────────────────────┐
                    │      Open airway         │──────────► ┌────────────────────┐
                    │  Not breathing normally  │            │ Send or go for AED  │
                    └─────────────────────────┘            │     Call 112*       │
                                 │                          └────────────────────┘
                                 ▼                          * or national emergency number
                    ┌─────────────────────────┐
                    │       CPR 30:2           │
                    │   Until AED is attached  │
                    └─────────────────────────┘
                                 │
                                 ▼
                           ◄ AED assesses ►
                              rhythm

      Shock advised    ◄────────►    No shock advised

         │                               │
         ▼                               ▼
      1 Shock                   Immediately resume:
         │                          CPR 30:2
         ▼                          for 2 min
   Immediately resume:
      CPR 30:2
      for 2 min

   Continue until the victim starts
   to move, opens eyes and to breathe normally
```
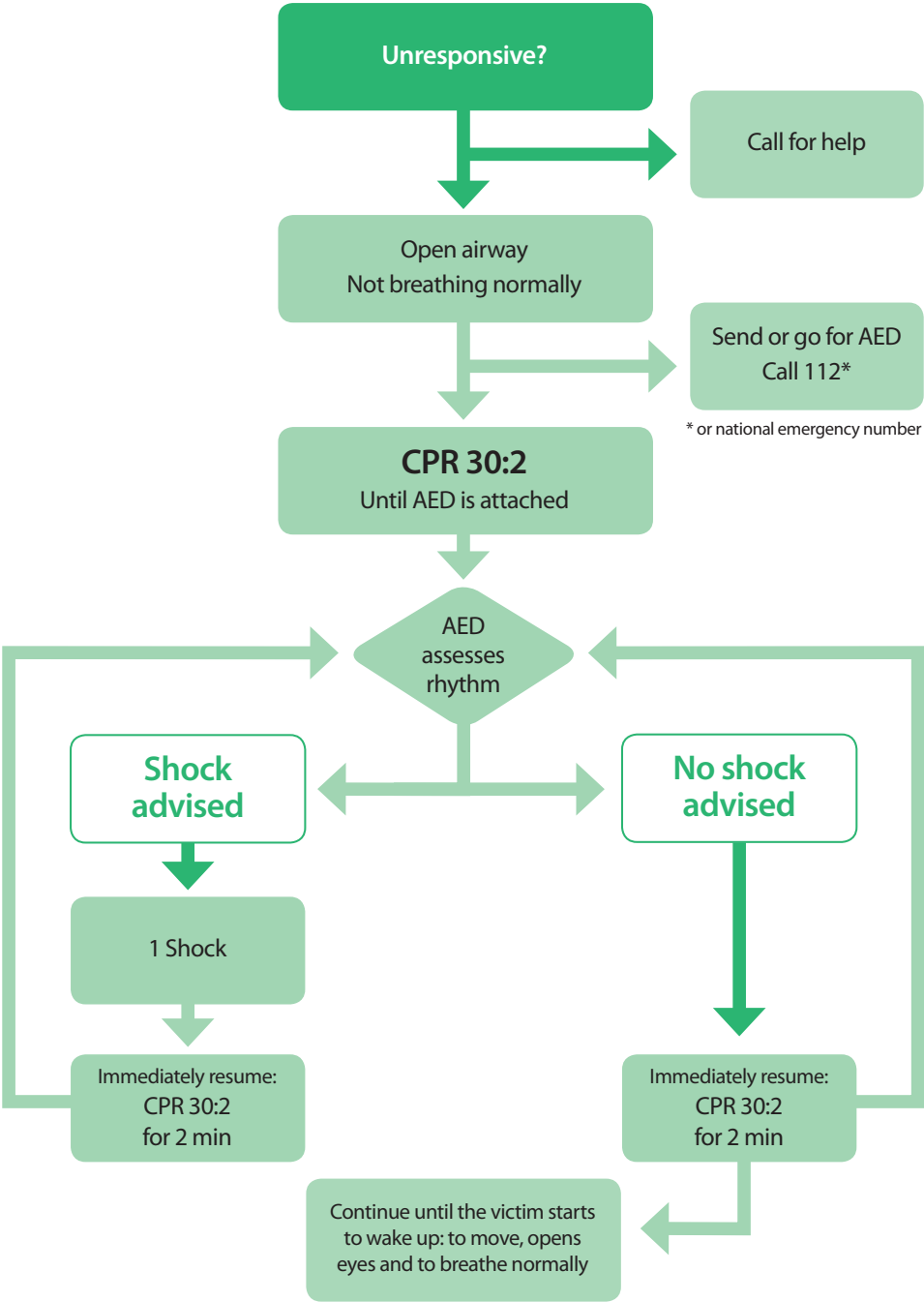
5.4. Sequence for use of an AED or shock-advisory defibrillator

1. Make sure the victim, any bystanders, and you are safe.

2. If the victim is unresponsive and not breathing normally:

    - Send someone for the AED and call for an ambulance or resuscitation team. If you are on your own, do both things yourself.

3. Start CPR according to the guidelines *(chapter 5)*.

4. As soon as the AED arrives:

    - Switch on the AED and attach the electrode pads. If more than one rescuer is present, continue CPR while this is done.

    - Follow the voice/visual directions.

    - Ensure that nobody touches the victim whilst the AED is analysing the rhythm.

5A. If a shock **IS** indicated:
    - Ensure that nobody touches the victim *(figure 9.6a)*.

    - Push the shock button *(figure 9.6b)* as directed.

    - Continue as directed by the voice/visual prompts.

5B. If **NO** shock is indicated:
    - Immediately resume CPR using a ratio of 30 compressions to 2 rescue breaths *(figure 9.6c)*.

    - Continue as directed by the voice/visual prompts.

6. Continue to follow the AED prompts until:
    - Qualified help (e.g. ambulance or resuscitation team) arrives and takes over.

    - The victim starts to breathe normally, or

    - You become exhausted.

Notes
- The carrying case with the AED must contain some strong scissors for cutting through clothing and a disposable razor for shaving excessive chest hair in order to obtain good electrode contact.

- If ALS providers are using the AED, they should implement other ALS interventions (advanced airway, ventilation, IV access, drug delivery, etc.) according to local protocols.

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Figure 9.6
Operation of a shock-advisory defibrillator and efficient CPR

6.   Manual defibrillation

Manual defibrillators enable the operator to diagnose the rhythm and deliver a shock rapidly without having to wait for rhythm analysis. This minimises the interruption in chest compressions. Manual defibrillators often have additional functions, such as the ability to deliver synchronised shocks, and external pacing facilities. The main disadvantage of these devices is that the operator has to be skilled in ECG rhythm recognition; therefore, in comparison with AEDs, extra training is required.

6.1.   Sequence for use of a manual defibrillator

This sequence is an integral part of the advanced life support treatment algorithm in chapter 6.

1.   Confirm cardiac arrest-check for signs of life or if trained to do so, breathing and pulse simultaneously.

2.   Call resuscitation team.

3.   Perform uninterrupted chest compressions while applying self-adhesive defibrillation/monitoring pads *(figure 9.7)* - one below the right clavicle and the other in the V6 position in the midaxillary line.

4.   Plan actions before pausing CPR for rhythm analysis and communicate these to the team.

5.   Stop chest compressions not longer than 2 seconds to check rhythm, resume chest compressions immediately.

6. Confirm VF/pVT, if in doubt use a print out rhythm strip; the designated person selects the appropriate energy on the defibrillator (150-200 J biphasic for the first shock and 150-360 J biphasic for subsequent shocks) and presses the charge button *(figure 9.8)*.

7. While the defibrillator is charging, warn all rescuers other than the individual performing the chest compressions to "stand clear" and remove any oxygen delivery device as appropriate. Ensure that the rescuer giving the compressions is the only person touching the patient.

8. Once the defibrillator is charged, tell the rescuer doing the chest compressions to "stand clear"; when clear, give the shock.

9. Without reassessing the rhythm or feeling for a pulse, restart CPR using a ratio of 30:2, starting with chest compressions.

10. Continue CPR for 2 min; the team leader prepares the team for the next pause in CPR.

11. Pause briefly to check the monitor.

12. If VF/pVT, repeat steps 6-11 above and deliver a second shock.

13. If VF/pVT persists repeat steps 6-8 above and deliver a third shock. Without reassessing the rhythm or feeling for a pulse, resume CPR (CV ratio 30:2) immediately after the shock, starting with chest compressions.

14. If IV/IO access has been obtained, during the next 2 minutes of CPR give adrenaline 1 mg and amiodarone 300 mg.

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15. Repeat this 2 min CPR-rhythm/pulse check-defibrillation sequence if VF/pVT persists.

16. Give further adrenaline 1 mg IV after alternate shocks (i.e., in practice, this will be about once every two cycles of the algorithm).

17. If organised electrical activity is seen during the pause to check the monitor, feel for a pulse:

    a. If a pulse is present, start post-resuscitation care.

    b. If no pulse is present, continue CPR and switch to the non-shockable algorithm.

18. If asystole is seen, continue CPR and switch to the non-shockable algorithm.

Figure 9.7
Applying defibrillator pads
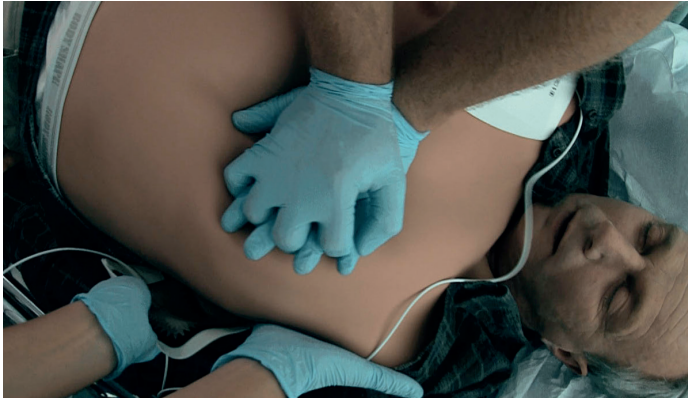
Figure 9.8
Charging during chest compressions

6.1.1.   Using handheld defibrillator paddles

As handheld paddles are still in use in many countries the following recommendations apply for their use:

6.a. Confirm VF, if in doubt use a print out rhythm strip; the designated person selects the appropriate energy on the defibrillator (maximum energy for monophasic devices), leave the paddles in the defibrillator and presses the charge button).

6.b. Apply conductive gel to the patients chest.

7. While the defibrillator is charging, warn all rescuers other than the individual performing the chest compressions to "stand clear" and remove any oxygen delivery device as appropriate. Ensure that the rescuer giving the compressions is the only person touching the patient.

8.a. Once the defibrillator is charged, tell the rescuer doing the chest compressions to "stand clear".

8.b. Move one of the charged paddles to the patients chest

8.c. Once safely positioned and kept in place move the second paddle to the patients chest.

8.d. Deliver shock and return both paddles to the defibrillator.

9. Without reassessing the rhythm or feeling for a pulse, restart CPR using a ratio of 30:2, starting with chest compressions.

7.   Pre-hospital defibrillation

There is evidence that performing chest compressions while retrieving and charging a defibrillator improves the probability of survival. EMS personnel should provide high-quality CPR while a defibrillator is retrieved, applied and charged. Defibrillation should not be delayed longer than needed to establish the need for defibrillation and charging. The routine delivery of a pre-specified period of CPR (e.g. two or three minutes) before rhythm analysis and a shock is delivered is not recommended.

Laypeople and first responders using AEDs should attach the device as soon as possible and follow the prompts.

8.   Synchronised cardioversion

If electrical cardioversion is used to convert atrial or ventricular tachyarrhythmias, the shock must be synchronised to occur with the R wave (not the T wave) of the electrocardiogram. By avoiding the relative refractory period, the risk of inducing VF is minimised. Most manual defibrillators incorporate a switch that enables the shock to be triggered by the R wave on the electrocardiogram. Electrodes are applied to the chest wall and cardioversion is achieved in the same way as attempted defibrillation but the operator must anticipate the slight delay between pressing the buttons and the discharge of the shock when the next R wave occurs. Do not move the defibrillator electrodes during this interval; otherwise the QRS complex will not be detected. The same safety precautions must be met as for attempted defibrillation.

Synchronisation can be difficult in VT because of the wide-complex and variable forms of ventricular arrhythmia. If synchronisation fails, give unsynchronised shocks to the unstable patient in VT to avoid prolonged delay in restoring sinus rhythm. Ventricular fibrillation or pulseless VT requires unsynchronised shocks. Conscious patients must be anaesthetised or sedated before attempting synchronised cardioversion.

Energy doses for cardioversion are discussed in chapter 11.

9. Cardiac pacemakers and implantable cardioverter- defibrillators

If the patient has a cardiac pacemaker or implantable cardioverter-defibrillator (ICD), be careful when placing the electrodes. Although modern pacemakers are fitted with protection circuits, the current may travel along the pacemaker wire or ICD lead causing burns where the electrode tip makes contact with the myocardium. This may increase impedance at the contact point and gradually increase the threshold for pacing. Place the defibrillator electrodes at least 8 cm from the pacemaker unit to minimise the risk. Alternatively place the pads in the antero-posterior or postero-lateral position as described above. If resuscitation is successful following defibrillation, check the pacemaker threshold regularly over the next two months. Recent case reports have documented rescuers receiving shocks from ICDs when in contact with the patient during CPR. It is particularly important to wear gloves and avoid skin-to-skin contact with the patient while performing CPR as there is no warning before the ICD discharges.

10. Internal defibrillation

Internal defibrillation using paddles applied directly across the ventricles requires considerably less energy than used for external defibrillation. Biphasic shocks are substantially more effective than monophasic shocks for direct defibrillation. For biphasic shocks, use 10-20 J, delivered directly to the myocardium through internal paddles. Monophasic shocks require approximately double these energy levels. Do not exceed 50 J when using internal defibrillation-failure to defibrillate at these energy levels requires myocardial optimisation before defibrillation is attempted again.

KEY LEARNING POINTS

- For the patient in VF/pVT, early defibrillation is the only effective means of restoring a spontaneous circulation.

- When using a defibrillator, minimise interruptions in chest compressions

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FURTHER READING

- Soar J, et al. European Resuscitation Council Guidelines for Resuscitation 2015. Section 3. Adult Advanced Life Support. 10.1016/j.resuscitation.2015.07.016; p99 - p146
- Soar J, Callaway CW, Aibiki M, et al. Part 4: Advanced life support: 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. 10.1016/j.resuscitation.2015.07.042; e71 - e122

Chapter 10.

Cardiac pacing

LEARNING OUTCOMES

To understand:

- the indications for cardiac pacing in the peri-arrest setting
- how to perform percussion pacing
- how to apply non-invasive, transcutaneous electrical pacing
- the problems associated with temporary transvenous pacing and how to correct them
- how to manage patients with implanted permanent pacemakers and cardioverter defibrillators in the setting of cardiac arrest and in the peri-arrest setting

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1.   Introduction

In some cardiac arrest or peri-arrest settings, appropriate use of cardiac pacing can be life-saving. Non-invasive pacing may be used to maintain cardiac output temporarily while expert help to deliver longer-term treatment is obtained. Non-invasive pacing can be established rapidly and is well within the capabilities of an ALS provider.

The ALS provider does not need to have a detailed technical knowledge of permanent cardiac pacemakers and implanted cardioverter defibrillators (ICDs) but needs to be able to recognise when one of these devices is present, when they are failing, and how the presence of an implanted device may influence the management of a cardiac arrest.

2.   The cardiac impulse - its formation and its failure

The electrical activity that stimulates each normal heartbeat arises in the sino-atrial (SA) node. This depolarises spontaneously and regularly without any external stimulus. Such behaviour is termed automaticity, and any cardiac tissue that possesses it is capable of initiating a heartbeat and behaving as the heart's natural pacemaker. Different parts of the conducting system depolarise spontaneously at different rates *(figure 10.1)*. The

fastest pacemaker will provide the cardiac rhythm and slower natural pacemakers will only take over if the faster ones fail. Examples may be seen in sinus arrest or extreme sinus bradycardia when the atrioventricular (AV) node may take over and provide a junctional escape rhythm, and in complete atrioventricular block (complete heart block-CHB) when the escape rhythm arises from the ventricular myocardium or from conducting tissue below the atrioventricular node.

Figure 10.1
Cardiac conducting system
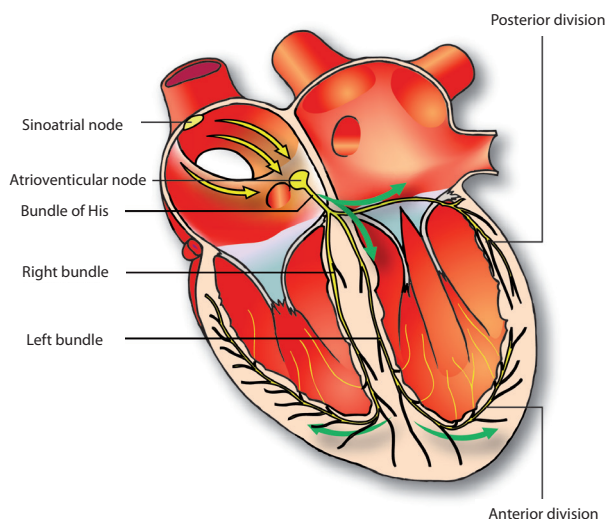
When CHB occurs at the level of the AV node, the most rapid automatic activity arises from cells immediately below the block and these become the new pacemaker. The intrinsic rate of these cells is relatively fast (often about 50 min^{-1}). The resulting escape rhythm is usually relatively stable and unlikely to fail and cause asystole.

The QRS complexes resulting from this type of block are narrow because the impulse is transmitted to the ventricles rapidly through an intact His-Purkinje system. This situation may be seen complicating acute inferior myocardial infarction. In this setting, narrow-complex CHB may not require pacing because the heart rate is often not especially slow and the risk of asystole is usually low.

Complete heart block can occur lower in the conducting system, for example, when all the fibres of the bundle branches are involved following anteroseptal myocardial infarction, or as a result of other disease including degenerative fibrosis and valve disease. Any automatic activity arising below this block in the distal Purkinje fibres is likely to be slow and unreliable. In this situation, the resulting QRS complexes will be broad, since the impulse passes slowly through ventricular muscle rather then rapidly through the His-Purkinje system. The

unreliable escape rhythm may fail briefly, leading to syncope (Stokes-Adams attack), or completely, causing ventricular standstill and cardiac arrest. Broad-complex CHB requires cardiac pacing, and the occurrence of significant ventricular pauses makes this urgent, as this implies a risk of asystole. The possible risk of more severe AV-block and asystole should always be considered in a patient who has presented with syncope and has any ECG evidence of conduction delay (e.g. long PR interval or bundle branch block). Such patients require at least cardiac monitoring and expert assessment.

In the peri-arrest setting, artificial pacemakers are used when the cardiac rhythm is unduly slow or unreliable and not responding to the treatment described in the peri-arrest algorithm for bradycardia *(chapter 11)*. However, pacing will be successful only if the heart is able to respond to the pacing stimulus. In the setting of cardiac arrest the continued presence of P waves makes this more likely.

Pacing is very rarely successful in asystole in the absence of P waves and should not be attempted routinely in this situation.

The stimulus to the myocardium may be either mechanical, as in percussion pacing, or electrical as in transcutaneous and transvenous pacing.

If a pacing stimulus induces an immediate QRS complex this is referred to as 'capture'. Always check that electrical activity seen on the ECG is accompanied by mechanical activity producing a palpable pulse.

3.   Methods of pacing

Methods of pacing are classified as:

Non-invasive
- percussion pacing ('fist pacing')
- transcutaneous pacing

Invasive
- temporary transvenous pacing
- permanent pacing with an implanted pacemaker

Implanted devices that deliver pacing include pacemakers implanted for the treatment of bradycardia, biventricular pacemakers implanted for the treatment of heart failure (cardiac resynchronisation therapy) and implanted cardioverter defibrillators (ICDs) that also have a pacemaker function.

3.1.   Non-invasive pacing

3.1.1. Percussion pacing

When bradycardia is so profound that it causes clinical cardiac arrest, percussion pacing can be used in preference to CPR because it is capable of producing an adequate cardiac output

with minimal trauma to the patient. It is more likely to be successful when ventricular standstill is accompanied by continuing P wave activity *(chapter 8)*.

3.1.2. How to perform percussion pacing

- With the side of a closed fist deliver repeated firm blows to the precordium lateral to the lower left sternal edge.

- Raise the hand about 10 cm above the chest for each blow.

- If initial blows do not produce a QRS complex try using slightly harder blows and try moving the point of contact around the precordium until a site is found that produces repeated ventricular stimulation.

Percussion pacing is not as reliable as electrical pacing in stimulating QRS complexes. If percussion does not produce a pulsed rhythm promptly, regardless of whether or not it stimulates QRS complexes, start CPR immediately.

Like CPR, percussion pacing is an emergency measure that is used to try to maintain circulation to vital organs and enable either recovery of a spontaneous cardiac rhythm or transcutaneous or transvenous pacing. Expert help must be sought as this is not a long-term solution.

3.2. Transcutaneous pacing

Compared with transvenous pacing, non-invasive transcutaneous pacing has the following advantages:

- It can be established very quickly.

- It is easy to perform and requires a minimum of training.

- It can be initiated by nurses, paramedics and doctors, while waiting for expert help to establish transvenous pacing.

The major disadvantage of transcutaneous pacing in the conscious patient is discomfort. The pacing impulse stimulates painful contraction of chest wall muscles as well as causing some direct discomfort. Treat conscious patients with iv analgesia and/or sedation. Many defibrillators incorporate a facility for transcutaneous pacing and the availability of multifunction, adhesive electrode pads capable of ECG monitoring, pacing, cardioversion, and defibrillation have made these units particularly versatile. Stand-alone non-invasive pacing devices may also be available in some hospital departments.

Most modern transcutaneous pacing systems are capable of demand pacing: intrinsic QRS complexes are sensed and pacing stimuli delivered only when needed. Be aware that additional simultaneous monitoring of the patient's rhythm by usual (small) ECG electrodes is necessary for most of the devices to function appropriately.

3.2.1. How to perform transcutaneous pacing

- Avoid any unnecessary delay in starting pacing, but pay careful attention to technique to increase the chance of success.

- Using scissors or a razor, quickly remove excess chest hair from the skin where the electrode pad is to be applied.

- Make sure that the skin is dry.

- Attach ECG monitoring electrodes and leads if necessary - these are needed with some transcutaneous pacing devices.

- Position the electrode pads in the conventional right pectoral-apical positions if possible *(figure 10.2a)*. If this is prevented (e.g. by chest trauma, pacemaker, or ICD implant) anterior-posterior (A-P) positions can be used *(figure 10.2b-d)*.

- If you are using a pacing device that is not capable of defibrillation, use A-P positions for the pacing electrodes so that defibrillator pads can still be used in the 'conventional' right pectoral and apical positions if cardiac arrest occurs.

- For right pectoral-apical positions place one electrode over the right pectoral muscle, just below the clavicle. Place the apical pad in the mid-axillary line, overlying the V6 ECG electrode position. It is important that this electrode is placed sufficiently laterally. Apply this pad to the chest wall, not over any breast tissue.

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Figure 10.2a
Pectoral-apical pad positions for external pacing

Figure 10.2b-d.
Anterior-posterior (AP) pad positions for external pacing.

For A-P positions place the anterior electrode on the left anterior chest wall, beside the sternum, overlying the V2 and V3 ECG electrode positions. Place the posterior electrode between the lower part of the left scapula and the spine, at the same horizontal level on the trunk as the anterior electrode.

- Different transcutaneous pacing devices have different properties. For example some require the operator to increase the current delivered with each pacing stimulus until electrical capture is achieved, whilst others use a constant current that cannot be adjusted and longer pulse duration (duration of the pacing stimulus) than

other devices. Make sure that you are familiar with the operation of the device that you are using.

- Most transcutaneous pacing devices offer pacing in demand mode; the pacemaker will be inhibited if it detects a spontaneous QRS complex. However, if there is a lot of movement artefact on the ECG this may inhibit the pacemaker. Avoid movement artefact as far as possible. If artefact still appears to be inhibiting the pacemaker, switch to fixed-rate pacing mode.

- Select an appropriate pacing rate. This will usually be in the range of 60-90 min^{-1} for adults, but in some circumstances (for example complete AV-block with an idioventricular rhythm at 50 min^{-1}) a slower pacing rate of 40 or even 30 min^{-1} may be appropriate to deliver pacing only during sudden ventricular standstill or more extreme bradycardia.

- If the pacing device has an adjustable energy output set this at its lowest value and turn on the pacemaker. Gradually increase the output while observing the patient and the ECG. As the current is increased the muscles of the chest wall will contract with each impulse and a pacing spike will appear on the ECG *(figure 10.3a)*. Increase the current until each pacing spike is followed immediately by a QRS complex, indicating electrical capture (typically with a current of 50-100 mA using a device with adjustable output). This means that the pacing stimuli are causing depolarisation of the ventricles *(figure 10.3b)*.

- Check that the apparent QRS complex is followed by a T wave. Occasionally, artefact generated by the pacing current travelling through the chest may be mistaken for a QRS complex, but such artefact will not be followed by a T wave *(figure 10.3a)*.

- If the highest current setting is reached and electrical capture has not occurred, try changing the electrode positions. Continued failure to achieve electrical capture may indicate non-viable myocardium, but other conditions (e.g. hyperkalaemia) may prevent successful pacing.

Having achieved electrical capture with the pacemaker, check for a pulse. A palpable pulse confirms the presence of a mechanical response of the heart to the paced QRS complex (i.e. contraction of the myocardium). Absence of a pulse in the presence of good electrical capture constitutes pulseless electrical activity (PEA). The most likely cause is severe myocardial failure but consider other possible causes of PEA in these circumstances.

Conscious patients usually experience considerable discomfort during transcutaneous pacing. Warn patients in advance that this may happen. They will often require intravenous analgesia and/or sedation if prolonged transcutaneous pacing is necessary. Reassess the patient frequently (ABCDE) because analgesics as well as sedative drugs may suppress the patient's respiratory effort.

When defibrillating a patient who has pacing-only electrode pads in place, apply the defibrillator paddles at least 2-3 cm from the pacing electrodes to prevent arcing of the defibrillation current.

Chest compressions can be given and other manual contact with the patient maintained as necessary with transcutaneous electrodes in place. There is no hazard from transcutaneous pacing to other people who are in contact with the patient. However, there is no benefit in trying to deliver transcutaneous pacing during chest compressions, so it is best to turn off the pacemaker whilst CPR is in progress.

When transcutaneous pacing produces an adequate cardiac output seek expert help immediately to decide about a transvenous pacing system.

Figure 10.3a
Transcutaneous pacing. Appearance of pacing spikes on ECG

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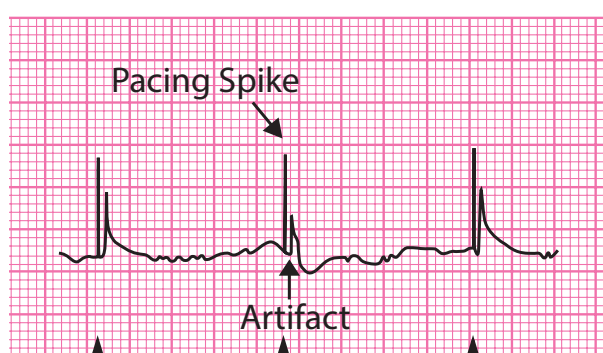
3.4. Invasive pacing

3.4.1. Temporary transvenous pacing

It is rare to have to attempt to insert a transvenous pacing wire during a cardiac arrest. In this setting, use non-invasive pacing to attempt to establish a cardiac output, and then seek expert help to establish transvenous pacing.

Failure of an existing temporary transvenous pacing system may cause cardiac arrest, particularly when the patient is pacing-dependent. Temporary transvenous pacing systems can fail in three ways:

- **High threshold**

When a temporary pacing lead is inserted the usual aim is to position its tip in the apex of the right ventricle, where it is least likely to be displaced. After positioning the lead, it is used to pace the heart and the voltage delivered by the pacemaker is decreased and increased to determine the minimum voltage needed to stimulate the ventricle. This is termed the pacing threshold and the usual aim is to achieve a threshold of < 1 V at the time of lead insertion. Higher thresholds suggest that the electrode is not making satisfactory contact with the myocardium, and the lead may need to be repositioned.

It is usual to pace the heart with a 3-4 V stimulus, well above the initial pacing threshold. Over the first days and weeks after insertion of a pacing lead (temporary or permanent) a rise in the threshold can be expected.

Check the threshold on temporary pacing leads at least daily to make sure that the output of the pacemaker is well above the threshold. If not, loss of capture may occur. This is seen on the ECG as a pacing spike without a subsequent QRS complex. Loss of capture may be intermittent, so any apparent 'missed beat' of this nature should prompt a repeat check of the pacing threshold.

If loss of capture occurs because of a high threshold, increase the output of the pacemaker immediately to well above the threshold. A sudden increase in pacing threshold may be caused by lead displacement, so obtain prompt expert help, as repositioning of the lead may be required.

- **Loss of electrical continuity**

Modern temporary transvenous pacing leads are bipolar. One electrode is at the tip of the lead and the second is about 1 cm proximal to the tip. Each electrode is connected by the lead to separate connectors at the other end, outside the patient. These are usually inserted into sockets at one end of a connecting cable that in turn is connected to the terminals of the pacemaker.

Make sure that all connections between the lead and the pacemaker are making good secure contact that is unlikely to be lost easily, for example by minor movement of the lead or cable.

Loss of contact at any point will stop delivery of the pacing stimulus to the heart, seen on the ECG as absence of a pacing spike. This may be intermittent and symptomless, or may be sudden and total and may cause syncope or cardiac arrest in asystole. When pacing failure is accompanied by loss of the pacing spike on the ECG, check all connections immediately; check that the pacemaker has not been turned off inadvertently and check that its batteries are not depleted. If no such cause is present another possible explanation is a fracture of a wire within its insulation. This usually causes intermittent pacing failure and the fracture is more likely to be in the connecting cable than in the pacing lead. If this is suspected change the connecting cable immediately.

- **Electrode displacement**

The tip of an endocardial transvenous pacing lead is usually positioned in the apex of the right ventricle. There should be enough slack in the lead as it passes through the right atrium to allow for changes in posture and deep inspiration, but not so much as to encourage displacement of the lead tip.

The tip of a pacing lead may also perforate the wall of the right ventricle and enter the pericardium with little or no apparent change in position on chest X-ray. Very rarely, this may cause pericardial tamponade, so consider this possibility if a patient with a recently implanted pacing lead suffers cardiac arrest with pulseless electrical activity.

When displacement or perforation occurs, the ECG will still show a pacing spike, but there is likely to be intermittent or complete loss of capture of the pacing stimulus, so the pacing spikes are not followed consistently by QRS complexes. When a pacing lead displaces but remains in the right ventricle it may trigger ventricular extrasystoles or more serious ventricular arrhythmia, including VT and VF. When transvenous pacing fails, there is a risk of ventricular standstill. This may be relatively short-lived and cause syncope, or prolonged and cause cardiac arrest in asystole. In this situation use non-invasive pacing until effective transvenous pacing has been re-established.

3.4.2. Implanted permanent pacing systems

Problems with permanent pacing systems are rare, because the connections between pacing electrodes and the pacemaker are much more secure. Occasional fracture of a permanent pacing lead may occur, usually following trauma such as a fall on to an outstretched arm on the side of the pacemaker. This may cause permanent or intermittent loss of the pacing spike.

When assessing a patient using the ABCDE approach check (during 'E') for the presence of an implanted device. These devices are usually implanted below the clavicle, often but not always on the left side. If a device is identified consider whether it is a pacemaker or an ICD and in the case of a pacemaker try to establish whether it was implanted as treatment for bradyarrhythmia or as treatment for heart failure.

If a patient with an implanted pacemaker or ICD has a cardiac arrest or requires cardioversion, place defibrillation pads at least 8 cm from the device. Devices that are implanted below the left clavicle usually present no problem with the use of standard defibrillator paddle positions. If a device has been implanted below the right clavicle, use A-P positions for defibrillation or cardioversion if possible. This is most easily and safely achieved using self-adhesive electrode pads rather than hand-held defibrillator paddles.

- **Biventricular pacing systems**

Until relatively recently, the usual reason for implantation of a permanent pacemaker has been the treatment of bradycardia, caused mostly by malfunction of the sino-atrial node or atrioventricular conduction. In recent years there has been increasing use of biventricular pacemakers as 'cardiac resynchronisation therapy' in patients with heart failure. Most of these patients do not require pacing for bradycardia. Pacing the apex of the right ventricle and the lateral wall of the left ventricle simultaneously improves the co-ordination of left ventricular contraction. These pacemakers require the same precautions during defibrillation and cardioversion as any other pacemaker, but failure of a pacemaker that has been inserted for this purpose will not usually cause any major change in heart rate or any dangerous rhythm abnormality.

4.   Implantable cardioverter-defibrillators

These devices resemble large implanted pacemakers. Many of them can function as demand pacemakers in the event of bradycardia and some will also deliver biventricular pacing

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for heart failure, as well as delivering defibrillation if required. National and international guidelines define indications for the implantation of an ICD, but accumulating evidence for improved survival after major myocardial infarction and in patients with heart failure has increased the use of these devices. Unlike a simple pacemaker, the primary function of an ICD is to terminate a life-threatening tachyarrhythmia. A 'simple' ICD can deliver a defibrillatory shock when it detects VF or very fast VT. More sophisticated devices can be programmed also to deliver critically timed pacing stimuli to attempt to terminate VT that is not especially fast and is unlikely to cause cardiac arrest, resorting to defibrillation only if the VT accelerates or degenerates into VF.

ICDs are implanted usually in the pectoral region in a similar position to pacemakers. Though these devices may seem complex, the means by which they sense changes in cardiac rhythm is relatively simple, depending mainly upon detection of rapid heart rates. Consequently, ICDs will occasionally misdiagnose an arrhythmia, or misinterpret other electrical signals, and deliver inappropriate shocks, which are very unpleasant for a conscious patient. Implantable cardioverter defibrillators can be disabled temporarily by holding or taping a magnet on the skin over the device. Seek expert help if ICD malfunction is suspected, as it may require reprogramming.

If a patient with an ICD has a cardiac arrest that is not terminated by the ICD, deliver CPR in the usual way. Until recently, it was thought that chest compressions could be undertaken without risk to the rescuer, even if the ICD delivers an internal shock to the patient during chest compression. However, there have been rare reports of rescuers receiving shocks from an ICD. This risk is minimised by wearing gloves. If a shockable cardiac arrest rhythm is present and is not terminated by the ICD, use external defibrillation in a standard fashion, taking the same precautions with choice of defibrillator paddle positions as in a patient with an implanted pacemaker. Assessment and checking of the device (ICD, pacemaker) by an expert is mandatory after external defibrillation or resuscitation as soon as possible.

Consider the possible requirement for ICD implantation in any patient who has been resuscitated from cardiac arrest in a shockable rhythm outside the context of proven acute ST segment elevation myocardial infarction. All such patients should be referred before discharge from hospital for assessment by a cardiologist with expertise in heart rhythm disorders.

KEY LEARNING POINTS

- Non-invasive pacing can be delivered by any ALS provider and is the immediate treatment for bradyarrhythmia that is a potential risk to the patient who does not respond to initial drug treatment.

- Non-invasive pacing is a temporary measure to be used until either a stable and effective spontaneous rhythm returns, or a competent person establishes transvenous pacing.

- Special precautions are necessary during resuscitation attempts in patients with implanted pacemakers and ICDs.

- The possible need for an ICD should be considered in patients resuscitated from cardiac arrest in VT or VF, in whom there is a risk of recurrence.

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Chapter 11.

Peri-arrest arrhythmias

> **LEARNING OUTCOMES**
>
> **To understand:**
>
> • the importance of arrhythmias that may precede or follow a cardiac arrest
>
> • how to assess peri-arrest arrhythmias
>
> • the principles of treatment of peri-arrest arrhythmias

1.   Introduction

Rhythm abnormalities that occur in the peri-arrest period may be considered in two main categories:

- **Arrhythmias that may lead to cardiac arrest** - many rhythm abnormalities occur without causing cardiac arrest: they are a relatively common complication of acute myocardial infarction (AMI) but are also common in patients with other cardiac abnormalities and in people who do not have coronary disease or structural heart disease. Untreated, some of these arrhythmias may lead to cardiac arrest or to avoidable deterioration in the patient's condition. Others may require no immediate treatment.

- **Arrhythmias that occur after initial resuscitation from cardiac arrest** - these often indicate that the patient's condition is still unstable and that there is a risk of deterioration or further cardiac arrest.

You should be able to recognise common arrhythmias and to know how to assess whether or not they require immediate treatment. The treatment algorithms described in this section have been designed to enable the non-specialist advanced life support (ALS) provider to treat a patient effectively and safely in an emergency; for this reason they have been kept as simple as possible. If patients are not acutely ill there may be treatment options, including the use of drugs (oral or parenteral) that will be less familiar to the non-expert. In this situation you should, whenever possible, seek advice from cardiologists or other senior doctors with the appropriate expertise.

2.   Sequence of actions

When an arrhythmia is present or suspected, start by assessing the patient using the ABCDE approach, including early establishment of cardiac monitoring *(see chapter 8)*. Assess the patient specifically for adverse features *(see below)*. Insert an intravenous cannula and, if appropriate, give oxygen. Whenever possible, record a 12-lead ECG at the earliest opportunity. This will help to identify the precise rhythm, either before treatment or retrospectively, if necessary with the help of an expert. Clinical assessment is of limited value in identifying the precise rhythm abnormality.

When you assess any patient with an arrhythmia address two factors:

1. the condition of the patient (presence or absence of adverse features)

2. the nature of the arrhythmia

3.   Adverse features

The presence or absence of adverse signs or symptoms will dictate the urgency and choice of treatment for most arrhythmias. The following adverse features indicate that a patient is unstable and at risk of deterioration, wholly or partly because of the arrhythmia:

- Shock-hypotension (systolic blood pressure < 90 mmHg), pallor, sweating, cold extremities, confusion or impaired consciousness.

- Syncope-transient loss of consciousness because of global reduction in blood flow to the brain.

- Heart failure-pulmonary oedema and/or raised jugular venous pressure (with or without peripheral oedema and liver enlargement).

- Myocardial ischaemia-typical ischaemic chest pain and/or evidence of myocardial ischaemia on a 12-lead ECG.

- Extremes of heart rate - in addition to the above adverse features it may be appropriate to consider extremes of heart rate as adverse signs in themselves, requiring more urgent assessment and treatment than less extreme tachycardia or bradycardia with no adverse signs.

  1. Extreme tachycardia: when heart rate increases diastole is shortened to a greater degree than systole. Rhythm abnormalities that cause very fast heart rates (e.g. > 150 min^{-1}) reduce cardiac output dramatically (because diastole is very short and the heart does not have time to fill properly) and reduce coronary blood flow (because this mostly occurs during diastole), potentially causing myocardial ischaemia. The faster the heart rate, the less well it will be tolerated.

  2. Extreme bradycardia: in general, the slower the bradycardia the less well it will be tolerated and heart rates below 40 min^{-1} are often tolerated poorly. This is especially so when people have severe heart disease and cannot compensate for the bradycardia by increasing stroke volume. Some people with very severe heart disease require faster than normal heart rates to maintain cardiac output, and even a 'normal' heart rate may be inappropriately slow for them.

4.   Treatment options

Depending on the clinical status of the patient (i.e. the presence or absence of adverse features) and the nature of the arrhythmia, immediate treatments can be categorised under five headings:

1. eliminate and/or correct relevant triggers like ischaemia, hypoxia, acidaemia, hypo-, hyperkalaemia, drugs, stress and pain

2. electrical (cardioversion for tachyarrhythmia or pacing for bradyarrhythmia)

3. simple clinical intervention (e.g. vagal manoeuvres, percussion pacing)

4. pharmacological (drug treatment)

5. no treatment needed

Most drugs act more slowly and less reliably than electrical treatments, so electrical treatment is usually the preferred treatment for an unstable patient with adverse features. When treating patients with absence of adverse features primarily with drugs be aware of possible deterioration either due to the drugs or by natural course of the arrhythmia. Be prepared for immediate electrical treatment (defibrillation, cardioversion or pacing).

If a patient develops an arrhythmia as a complication of some other condition (e.g. infection, AMI, heart failure), make sure that the underlying condition is assessed and treated appropriately, involving relevant experts if necessary.

5.   Subsequent monitoring and treatment

After successful treatment of an arrhythmia continue to monitor the patient until you are confident that the risk of further arrhythmia is low. Remember always to record a 12-lead ECG **after** successful treatment of an arrhythmia because this may show abnormalities (or absence of abnormalities) that will be important in planning future management. Correct all reversible factors that may predispose to further arrhythmia. Ensure that appropriate further expert help and advice is obtained at the most appropriate time for the patient.

6.   Tachycardia

6.1.   If the patient has adverse features

If the patient is unstable and deteriorating, with any of the adverse signs and symptoms described above being caused by the tachycardia, attempt synchronised cardioversion immediately *(figure 11.1)*. In patients with otherwise normal hearts, serious signs and symptoms are uncommon if the ventricular rate is < 150 beats min^{-1}. Patients with impaired cardiac function or significant comorbidity may be symptomatic and unstable at lower heart rates. If cardioversion fails to restore sinus rhythm and the patient remains unstable, give amiodarone 300 mg intravenously over 10-20 minutes and re-attempt electrical cardioversion. The loading dose of amiodarone can be followed by an infusion of 900 mg over 24 hours.

Repeated attempts of electrical cardioversion are not appropriate for recurrent (within hours or days) paroxysms (self-terminating episodes) of atrial fibrillation. This is relatively common in critically ill patients who may have ongoing precipitating factors causing the arrhythmia (e.g. metabolic disturbance, sepsis). Cardioversion does not prevent subsequent arrhythmias. If there are recurrent episodes, treat them with drugs.

6.2.   Synchronised electrical cardioversion

Carry out cardioversion under general anaesthesia or analgesia/conscious sedation, administered by a healthcare professional competent in the technique being used.

Ensure that the defibrillator is set to deliver a synchronised shock. This delivers the shock to coincide with the R wave. An unsynchronised shock could coincide with a T wave and cause ventricular fibrillation (VF).

For a broad-complex tachycardia or atrial fibrillation, start with 120-150 J biphasic shock (200 J monophasic) and increase in increments if this fails. Atrial flutter and regular narrow-complex tachycardia will often be terminated by lower-energy shocks: start with 70-120 J biphasic (100 J monophasic). For atrial fibrillation and flutter use anteroposterior defibrillator pad positions when it is practicable to do so.

When delivering the shock, press the shock button and keep it pressed until after the shock has occurred - there may be a slight delay before the shock is delivered.

If a second shock is needed, reactivate the synchronisation switch if necessary.

6.3.   If the patient has no adverse features

If the patient with tachycardia is stable (no adverse signs or symptoms) and is not deteriorating, pharmacological treatment may be possible. Evaluate the rhythm using a 12-lead ECG and assess the QRS duration. If the QRS duration is equal or greater than 0.12 seconds (3 small squares on standard ECG paper) it is classified as a broad complex tachycardia. If the QRS duration is less than 0.12 seconds it is a narrow complex tachycardia.

All anti-arrhythmic treatments-physical manoeuvres, drugs, or electrical treatment- can also be pro-arrhythmic, so that clinical deterioration may be caused by the treatment rather than lack of effect. The use of multiple anti-arrhythmic drugs or high doses of a single drug can cause myocardial depression and hypotension. This may cause a deterioration of the cardiac rhythm and patients condition. Expert help should be sought before using repeated doses or combinations of anti-arrhythmic drugs.

Figure 11.1
Tachycardia algorithm

- Assess using the ABCDE approach
- Give oxygen if appropriate and obtain IV access
- Monitor ECG, BP, SpO$_2$, record 12 lead ECG
- Identify and treat reversible causes (e.g. electrolyte abnormalities)

Assess for evidence of adverse signs
1. Shock   3. Myocardial ischaemia
2. Syncope   4. Heart failure

Unstable →

Synchronised DC Shock*
Up to 3 attempts

- Amiodarone 300 mg IV over 10-20 min and repeat shock; followed by:
- Amiodarone 900 mg over 24 h

Stable

Is QRS narrow (< 0.12 sec)?

Broad

Narrow

Broad QRS
Is QRS regular?

Narrow QRS
Is rhythm regular?

Irregular   Regular

Regular   Irregular

Seek expert help ⚠

- Use vagal manoeuvres
- Adenosine 6 mg rapid IV bolus; if unsuccessful give 12 mg; if unsuccessful give further 12 mg.
- Monitor ECG continuously

Irregular Narrow Complex Tachycardia
Probable atrial fibrillation
Control rate with:
- β-Blocker or diltiazem
- Consider digoxin or amiodarone if evidence of heart failure
Anticoagulate if duration > 48h

Normal sinus rhythm restored?   NO   Seek expert help ⚠

YES

Possibilities include:
- **AF with bundle branch block** treat as for narrow complex
- **Polymorphic VT** (e.g. torsades de pointes - give magnesium 2 g over 10 min)

If Ventricular Tachycardia
(or uncertain rhythm):
- Amiodarone 300 mg IV over 20-60 min; then 900 mg over 24 h
- If previously confirmed **SVT with bundle branch block:** Give adenosine as for regular narrow complex tachycardia

Probable re-entry PSVT:
- Record 12-lead ECG in sinus rhythm
- If recurs, give adenosine again & consider choice of anti-arrhythmic prophylaxis

Possible **atrial flutter**
- Control rate (e.g. ß-Blocker)

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*Attempted electrical cardioversion on conscious patients is always undertaken under sedation or general anaesthesia

6.4. Broad-complex tachycardia

Broad-complex tachycardias are usually ventricular in origin. Although broad-complex tachycardias may be caused by supraventricular rhythms with aberrant conduction, in the unstable patient in the peri-arrest context assume they are ventricular in origin. In the stable patient with broad-complex tachycardia, the next step is to determine if the rhythm is regular or irregular.

6.4.1. Regular broad-complex tachycardia

A regular broad-complex tachycardia is likely to be ventricular tachycardia or SVT with bundle branch block. If there is uncertainty about the source of the arrhythmia, give intravenous adenosine as it may convert the rhythm to sinus and help diagnose the underlying rhythm.

Stable ventricular tachycardia can be treated with amiodarone 300 mg intravenously over 20-60 minutes followed by an infusion of 900 mg over 24 hours. Specialist advice should be sought before considering alternatives treatments such as procainamide or sotalol.

6.4.2. Irregular broad-complex tachycardia

Irregular broad complex tachycardia is most likely to be AF with bundle branch block. Another possible cause is AF with ventricular pre-excitation (Wolff–Parkinson–White (WPW) syndrome). In this case there is more variation in the appearance and width of the QRS complexes than in AF with bundle branch block. A third possible cause is polymorphic VT (e.g. torsades de pointes), although this rhythm is relatively unlikely to be present without adverse features.

Seek expert help with the assessment and treatment of irregular broad-complex tachyarrhythmia. If treating AF with bundle branch block, treat as for AF *(see below)*. If pre-excited AF (or atrial flutter) is suspected, avoid adenosine, digoxin, verapamil and diltiazem. These drugs block the AV node and cause a relative increase in pre-excitation – this can provoke more dangerous tachycardias. Electrical cardioversion is usually the safest treatment option.

Treat torsades de pointes VT immediately by stopping all drugs known to prolong the QT interval. Correct electrolyte abnormalities, especially hypokalaemia. Give magnesium sulphate, 2 g, intravenously over 10 minutes. Obtain expert help, as other treatment (e.g. overdrive pacing) may be indicated to prevent relapse once the arrhythmia has been corrected. If adverse features develop (which is usual), arrange immediate synchronised cardioversion. If the patient becomes pulseless, attempt defibrillation immediately (cardiac arrest algorithm).

6.5. Narrow-complex tachycardia

The first step in the assessment of a narrow complex tachycardia is to determine if it is regular or irregular.

The commonest regular narrow-complex tachycardias include:

- sinus tachycardia;

- AV nodal re-entry tachycardia (AVNRT, the commonest type of SVT);

- AV re-entry tachycardia (AVRT), which is associated with Wolff-Parkinson-White (WPW) syndrome;

- atrial flutter with regular AV conduction (usually 2:1).

Irregular narrow-complex tachycardia is most commonly AF or sometimes atrial flutter with variable AV conduction ('variable block').

6.5.1. Regular narrow-complex tachycardia

- **Sinus tachycardia**

Sinus tachycardia is a common physiological response to a stimulus such as exercise or anxiety. In a sick patient it may be seen in response to many stimuli, such as pain, fever, anaemia, blood loss and heart failure. Treatment is almost always directed at the underlying cause; trying to slow sinus tachycardia primarily using antiarrhythmic drugs will make the situation worse.

- **AVNRT and AVRT (paroxysmal supraventricular tachycardia)**

AVNRT is the commonest type of paroxysmal SVT, often seen in people without any other form of heart disease and is relatively uncommon in a peri-arrest setting. It causes a regular narrow-complex tachycardia, often with no clearly visible atrial activity on the ECG. Heart rates are usually well above the typical range of sinus rates at rest (60–120 beats min^{-1}). It is usually benign, unless there is additional co-incidental structural heart disease or coronary disease.

AV re-entry tachycardia (AVRT) is seen in patients with the WPW syndrome and is also usually benign unless there happens to be additional structural heart disease. The common type of AVRT is a regular narrow-complex tachycardia, also often having no visible atrial activity on the ECG.

- **Atrial flutter with regular AV conduction (often 2:1 block)**

Atrial flutter with regular AV conduction (often 2:1 block) produces a regular narrow-complex tachycardia in which it may be difficult to see atrial activity and identify flutter waves with confidence, so it may be indistinguishable initially from AVNRT and AVRT. When atrial flutter with 2:1 block or even 1:1 conduction is accompanied by bundle branch block, it produces a regular broad-complex tachycardia that will usually be very difficult to distinguish from VT. Treatment of this rhythm as if it is VT will usually be effective, or will lead to slowing of the ventricular response and identification of the rhythm. Most typical atrial flutter has an atrial rate of about 300 beats min^{-1}, so atrial flutter with 2:1 block tends to produce a tachycardia of about 150 beats min^{-1}. Much faster rates are unlikely to be due to atrial flutter with 2:1 block.

- **Treatment of regular narrow-complex tachyarrhythmia**

If the patient is unstable with adverse features caused by the arrhythmia, attempt

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synchronised electrical cardioversion. It is reasonable to give adenosine to an unstable patient with a regular narrow-complex tachycardia while preparations are made for synchronised cardioversion; however, do not delay electrical cardioversion if the adenosine fails to restore sinus rhythm. In the absence of adverse features, proceed as follows.

- Start with vagal manoeuvres: carotid sinus massage or the Valsalva manoeuvre will terminate up to a quarter of episodes of paroxysmal SVT. Carotid sinus massage stimulates baroreceptors, which increase vagal tone and reduces sympathetic drive, which slows conduction via the AV node. A Valsalva manoeuvre (forced expiration against a closed glottis) in the supine position may be the most effective technique. A practical way of achieving this without protracted explanation is to ask the patient to blow into a 20 ml syringe with enough force to push back the plunger. Record an ECG (preferably multi-lead) during each manoeuvre. If the rhythm is atrial flutter, slowing of the ventricular response will often occur and demonstrate flutter waves.

- If the arrhythmia persists and is not atrial flutter, use adenosine. Give 5-6 mg (depending on the preparation available) as a rapid intravenous bolus. Record an ECG (preferably multi-lead) during each injection. If the ventricular rate slows transiently but the arrhythmia then persists, look for atrial activity such as atrial flutter or other atrial tachycardia and treat accordingly. If there is no response to adenosine 5-6 mg, give a 10-12 mg bolus; if there is no response, give one further 10-12 mg bolus. This strategy will terminate 90-95 % of supraventricular arrhythmias.

- Successful termination of a tachyarrhythmia by vagal manoeuvres or adenosine indicates that it was almost certainly AVNRT or AVRT. Monitor the patients for further rhythm abnormalities. Treat recurrence either with further adenosine or with a longer-acting drug with AV nodal-blocking action (e.g. diltiazem or verapamil).

- If adenosine is contraindicated or fails to terminate a regular narrow-complex tachycardia without demonstrating that it is atrial flutter, give a calcium channel blocker (e.g. verapamil or diltiazem).

6.5.2. Rapid narrow-complex tachycardia with no pulse

Rarely, a very rapid (usually > 250 min^{-1}) narrow-complex tachycardia can impair cardiac output to such an extent that the pulse may be impalpable and consciousness impaired. If the patient is pulseless and unconscious this situation is pulseless electrical activity (PEA) and you should start CPR. As the arrhythmia is potentially treatable by DC shock the most appropriate treatment then is immediate synchronised cardioversion, so this is an exception to the non-shockable limb of the ALS algorithm *(chapter 6)*.

6.5.3. Irregular narrow-complex tachycardia

An irregular narrow-complex tachycardia is most likely to be AF with an uncontrolled ventricular response or, less commonly, atrial flutter with variable AV-block. Record a 12-lead ECG to identify the rhythm. If the patient is unstable with adverse features caused by the arrhythmia, attempt synchronised electrical cardioversion.

Do not use adenosine if the rhythm is obviously atrial fibrillation/flutter. If there are no adverse features, treatment options include:

- rate control by drug therapy
- rhythm control using drugs to encourage pharmacological cardioversion
- rhythm control by electrical cardioversion
- prevention of complications (e.g. anticoagulation)

Obtain expert help to determine the most appropriate treatment for the individual patient. The longer a patient remains in AF the greater is the likelihood of atrial thrombus developing. In general, patients who have been in AF for > 48 h should not be treated by cardioversion (electrical or chemical) until they have been fully anticoagulated for at least 3 weeks, or unless trans-oesophageal echocardiography has excluded the presence of atrial thrombi. If the clinical situation dictates that cardioversion is needed more urgently, give either regular low-molecular-weight heparin in therapeutic dose or an intravenous bolus injection of unfractionated heparin followed by a continuous infusion to maintain the activated partial thromboplastin time (APTT) at 1.5-2 times the reference control value. Continue heparin therapy and commence oral anticoagulation after successful cardioversion. Seek expert advice on the duration of anticoagulation, which should be a minimum of 4 weeks, often substantially longer.

7. Bradycardia

A bradycardia is defined as a heart rate of < 60 beats min^{-1}. Common causes for Bradycardia are:

- physiological (e.g. in athletes)
- cardiac (e.g. myocardial infarction; myocardial ischaemia; sick sinus syndrome)
- non-cardiac (e.g. vasovagal response, hypothermia; hypoglycaemia; hypothyroidism; raised intracranial pressure)
- drug toxicity (e.g. digoxin; beta blockers; calcium channel blockers).

Bradycardias are caused by reduced sinoatrial node firing or failure of the atrial-ventricular conduction system. Reduced sinoatrial node firing is seen in sinus bradycardia (caused by excess vagal tone), sinus arrest, and sick sinus syndrome. Atrioventricular (AV) blocks are divided into first, second, and third degrees and may be associated with multiple medications or electrolyte disturbances, as well as structural problems caused by acute myocardial infarction and myocarditis. A first-degree AV-block is defined by a prolonged P-R interval (> 0.20 s), and is usually benign. Second-degree AV-block is divided into Mobitz types I and II. In Mobitz type I, the block is at the AV node, is often transient and may be asymptomatic. In Mobitz type II, the block is most often below the AV node at the bundle of His or at the bundle branches, and is often symptomatic, with the potential to progress to complete AV-block. Third-degree heart block is defined by AV dissociation, which may be permanent or transient, depending on the underlying cause.

- **Initial assessment**

Assess the patient with bradycardia using the ABCDE approach. Consider the potential cause

of the bradycardia and look for the adverse signs. Treat any reversible causes of bradycardia identified in the initial assessment. If adverse signs are present start to treat the bradycardia. Initial treatment are pharmacological, with pacing being reserved for patients unresponsive to pharmacological treatment or with risks factors for asystole *(figure 11.2)*.

7.1.   Pharmacological treatment for bradycardia

If adverse signs are present, give atropine, 500 micrograms, intravenously and, if necessary, repeat every 3–5 minutes to a total of 3 mg. Doses of atropine of less than 500 micrograms, paradoxically, may cause further slowing of the heart rate. In healthy volunteers a dose of 3 mg produces the maximum achievable increase in resting heart rate. Use atropine cautiously in the presence of acute coronary ischaemia or myocardial infarction; increased heart rate may worsen ischaemia or increase the zone of infarction.

If treatment with atropine is ineffective, consider second line drugs. These include isoprenaline (5 micrograms min^{-1} starting dose), adrenaline (2-10 micrograms min^{-1}) and dopamine (2-10 micrograms kg^{-1} min^{-1}). Theophylline (100-200 mg slow intravenous injection) should be considered if the bradycardia is caused by inferior myocardial infarction, cardiac transplant or spinal cord injury. Consider giving intravenous glucagon if beta-blockers or calcium channel blockers are a potential cause of the bradycardia. Do not give atropine to patients with cardiac transplants - it can cause a high-degree AV-block or even sinus arrest.

7.2.   Cardiac pacing for bradycardia

Initiate transcutaneous pacing immediately if there is no response to atropine, or if atropine is unlikely to be effective.

Transcutaneous pacing can be painful and may fail to produce effective mechanical capture. Verify mechanical capture and reassess the patient's condition. Use analgesia and sedation to control pain, and attempt to identify the cause of the bradyarrhythmia.

If atropine is ineffective and transcutaneous pacing is not immediately available, fist pacing can be attempted while waiting for pacing equipment. Give serial rhythmic blows with the closed fist over the left lower edge of the sternum to pace the heart at a physiological rate of 50-70 beats min^{-1}.

Seek expert help to assess the need for temporary transvenous pacing. Temporary transvenous pacing should be considered if there are is a history of recent asystole; Mobitz type II AV-block; complete (third-degree) heart block (especially with broad QRS or initial heart rate < 40 beats min^{-1}) or evidence of ventricular standstill of more than 3 seconds.

7.3.   If the patient has no adverse features

In a patient with bradycardia and no adverse features or high risk of progression to asystole, do not initiate immediate treatment. Continue to monitor the patient. Assess the patient to identify the cause of the bradycardia. If the cause is physiological or reversible (e.g. by stopping suppressant drug therapy) no further treatment may be needed. Seek expert help to arrange appropriate further assessment and treatment for those with other causes of bradycardia.

Figure 11.2.
Bradycardia algorythm

- Assess using the ABCDE approach
- Give oxygen if appropriate and obtain IV access
- Monitor ECG, BP, SpO_2, record 12 lead ECG
- Identify and treat reversible causes (e.g. electrolyte abnormalities)

Assess for evidence of adverse signs

1. **Shock**
2. **Syncope**
3. **Myocardial ischaemia**
4. **Heart failure**

YES → NO

Atropine 500 mcg IV

Satisfactory response? — YES →

Risk of asystole?
- Recent asystole
- Mobitz II AV-block
- Complete heart block with broad QRS
- Ventricular pause > 3s

NO

Interim measures:
- Atropine 500 mcg IV repeat to maximum of 3 mg
- Isoprenaline 5 mcg min^{-1} IV
- Adrenaline 2-10 mcg min^{-1} IV
- Alternative drugs*

OR
- Transcutaneous pacing

← YES

NO

⚠ **Seek expert help**
Arrange transvenous pacing

Observe

***Alternatives include:**
- Aminophylline
- Dopamine
- Glucagon (if beta-blocker or calcium channel blocker overdose)
- Glycopyrrolate can be used instead of atropine

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KEY LEARNING POINTS

- Arrhythmias occurring after resuscitation from cardiac arrest and ROSC may need treatment to stabilise the patient and prevent recurrence of cardiac arrest.

- In other settings some arrhythmias may require prompt treatment to prevent deterioration, including progression to cardiac arrest, and others do not require immediate treatment.

- The urgency for treatment and the best choice of treatment is determined by the condition of the patient (presence or absence of adverse features) and by the nature and cause of the arrhythmia.

- Assessment of a patient with an arrhythmia should follow the ABCDE approach.

- Whenever possible the arrhythmia should be documented on a 12-lead ECG.

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Chapter 12.

Cardiac arrest in special circumstances

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LEARNING OUTCOMES

To understand how resuscitation techniques are modified in the special circumstances of:

- hypo-/hyperkalaemia and other electrolyte disorders
- poisoning
- accidental hypothermia
- hyperthermia
- drowning
- asthma
- anaphylaxis
- cardiac arrest following cardiac surgery
- trauma
- pregnancy
- obesity
- electrocution

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1. Introduction

Resuscitation needs to be modified in specific circumstances. Early recognition of signs and symptoms and effective treatment will often prevent cardiac arrest. These conditions account for a large proportion of cardiac arrests in younger patients with no co-existing disease. It is essential to ask for appropriate expert help early for most of these conditions as they will require specialist interventions.

Survival in all these conditions still relies on using the ABCDE approach to help prevent cardiac arrest. If cardiac arrest does occur, high-quality CPR with minimal interruption and treatment of reversible causes are still the most important interventions.

2. Hypo-/hyperkalaemia and other electrolyte disorders

Electrolyte abnormalities can cause cardiac arrhythmias or cardiorespiratory arrest. Life-threatening arrhythmias are associated most commonly with potassium disorders, particularly hyperkalaemia, and less commonly with disorders of serum calcium and magnesium.

Consider life-threatening electrolyte disturbances in patient groups at risk (e.g. renal failure, severe burns, heart failure and diabetes mellitus). Electrolyte values for definitions are quoted as a guide to clinical decision-making. The precise values that trigger treatment decisions will depend on the patient's clinical condition and rate of change of electrolyte values.

2.1. Prevention of electrolyte disorders

- Treat life-threatening electrolyte abnormalities before cardiac arrest occurs.

- Remove precipitating factors (e.g. drugs) and monitor electrolyte concentrations to prevent recurrence of the abnormality.

- Monitor renal function in patients at risk of electrolyte disorders (e.g. patients with chronic kidney disease, heart failure).

- Review renal replacement therapy (e.g. haemodialysis) regularly to avoid inappropriate electrolyte shifts during treatment.

2.2. Potassium disorders

Potassium homeostasis

Extracellular potassium concentration is regulated tightly between 3.5-5.0 mmol l^{-1}. A large concentration gradient normally exists between intracellular and extracellular fluid compartments. Evaluation of serum potassium must take into consideration the effects of changes in serum pH. When serum pH decreases (acidaemia), serum potassium concentration increases, because potassium shifts from the cellular to the vascular space. When serum pH increases (alkalaemia), serum potassium concentration decreases because potassium shifts into cells. Anticipate the effects of pH changes on serum potassium during therapy for hyperkalaemia or hypokalaemia.

2.2.1. Hyperkalaemia

Hyperkalaemia is the most common electrolyte disorder associated with cardiac arrest and usually caused by increased potassium release from cells or impaired excretion by the kidneys, drugs and metabolic acidosis.

Definition

There is no universal definition. We have defined hyperkalaemia as a serum potassium concentration > 5.5 mmol l^{-1}; in practice, hyperkalaemia is a continuum. As the potassium concentration increases, the risk of adverse events increases and the need for urgent treatment increases. Severe hyperkalaemia has been defined as a serum potassium concentration > 6.5 mmol l^{-1}.

Causes

The causes of hyperkalaemia include:

- renal failure (i.e. acute kidney injury or chronic kidney disease)

- drugs, e.g. angiotensin converting enzyme inhibitors (ACEI), angiotensin II receptor blockers (ARB), potassium sparing diuretics, non-steroidal anti-inflammatory drugs (NSAIDs), beta-blockers, trimethoprim

- tissue breakdown (rhabdomyolysis, tumour lysis, haemolysis)

- metabolic acidosis

- endocrine disorders (Addison's disease)

- diet (may be sole cause in patients with advanced chronic kidney disease)

- spurious - pseudo-hyperkalaemia (haematological disorders, prolonged transit time to the laboratory, poor storage conditions)

The risk of hyperkalaemia increases when there is a combination of causative factors such as the concomitant use of ACEI or ARB and potassium sparing diuretics.

Recognition of hyperkalaemia

Exclude hyperkalaemia in all patients with an arrhythmia or cardiac arrest. Patients can present with weakness progressing to flaccid paralysis, paraesthesia, or depressed deep tendon reflexes. The effect of hyperkalaemia on the ECG depends on the absolute serum potassium concentration as well as the rate of increase *(figure 12.1)*.

ECG changes with hyperkalaemia are usually progressive and include:

- first degree heart block (prolonged PQ or PR interval) (> 0.2 s)

- flattened or absent P waves

- tall, peaked (tented) T waves (T wave larger than R wave in more than one lead)

- ST-segment depression

- S and T wave merging (sine wave pattern)

- widened QRS (≥ 0.12 s)

- bradycardia (sinus bradycardia or AV-block)

- ventricular tachycardia

- cardiac arrest (VF/pVT, PEA, asystole)

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Most patients will have ECG abnormalities at a serum potassium concentration > 6.7 mmol l^{-1}. The use of a blood gas analyser that measures potassium helps reduce delays in recognition.

Figure 12.1
12-lead ECG showing features of hyperkalaemia
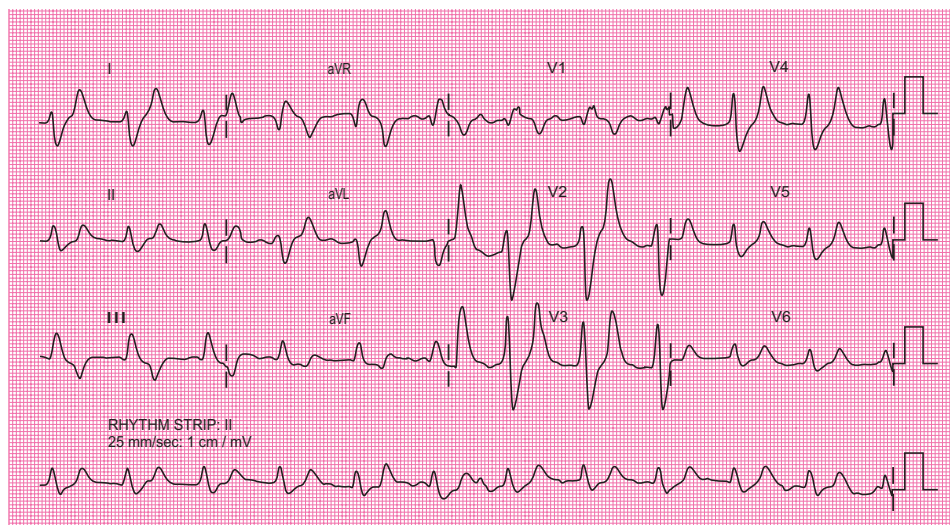
Treatment of hyperkalaemia

The five key treatment strategies for hyperkalaemia are:

1. cardiac protection

2. shifting potassium into cells

3. removing potassium from the body

4. monitoring serum potassium and blood glucose

5. prevention of recurrence of hyperkalaemia

When hyperkalaemia is strongly suspected, e.g. in the presence of ECG changes, start life-saving treatment even before laboratory results are available. Involve expert help from renal or intensive care teams at an early stage especially for those patients who might require renal replacement therapies (e.g. dialysis). Continue to monitor serum potassium for a minimum of 24 hours after an episode to avoid rebound hyperkalaemia.

Patient not in cardiac arrest

Assess ABCDE (Airway, Breathing, Circulation, Disability, Exposure) and correct any abnormalities. If hypovolaemic, give fluids to enhance urinary potassium excretion. Obtain intravenous access, check serum potassium and record an ECG. Treatment is determined according to severity of hyperkalaemia. Approximate values are provided to guide treatment. Follow the hyperkalaemia emergency treatment algorithm *(figure 12.2)*.

Figure 12.2
Hyperkalaemia emergency treatment algorithm

Assess using ABCDE approach
- Assess using ABCDE approach
- 12-lead ECG and monitor cardiac rhythm if serum potassium (K$^+$) ≥ 6.5 mmol L^{-1}
- Exclude pseudohyperkalaemia
- Give empirical treatment for arrhythmia if hyperkalaemia suspected

MILD
K$^+$ 5.5 - 5.9 mmol L^{-1}
Consider cause and need for treatment

MODERATE
K$^+$ 6.0 - 6.4 mmol L^{-1}
Treatment guided by clinical scenario, ECG and rate of rise

SEVERE
K$^+$ ≥ 6.5 mmol L^{-1}
Emergency treatment indicated

Seek expert help ⚠

ECG changes?
- Peaked T waves
- Flat / absent P waves
- Broad QRS
- Sine wave
- Bradycardia
- VT

NO   YES

Protect the heart

IV calcium
10 mL 10 % calcium chloride IV
OR 30 mL 10 % calcium gluconate IV
- Use large IV access and give over 5-10 min
- Repeat ECG
- Consider further dose after 5 min if ECG changes persist

Shift K$^+$ into cells

Insulin–glucose IV infusion
Glucose (25 g) with 10 units soluble insulin over 15 min IV
25 g glucose = 50 mL 50 % glucose OR 125 mL 20 % glucose

Risk of hypoglycaemia

Salbutamol 10-20 mg nebulised

Remove K$^+$ from body

Consider calcium resonium
15 g x 4/day oral or 30 g x 2/day per rectum

Consider dialysis
Seek expert help ⚠

Monitor K$^+$ and blood glucose

Monitor serum potassium and blood glucose

K$^+$ ≥ 6.5 mmol L^{-1} despite medical therapy

Prevention

Consider cause of hyperkalaemia and prevent recurrence

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Mild elevation (5.5-5.9 mmol l⁻¹):

- Address cause of hyperkalaemia to correct and avoid further rise in serum potassium (e.g. drugs, diet).

- If treatment is indicated, remove potassium from the body with potassium exchange resins: calcium resonium 15 to 30 g or sodium polystyrene sulfonate (Kayexalate) 15 to 30 g, given either orally or by retention enema (onset in > 4 hours).

Moderate elevation (6-6.4 mmol l⁻¹) without ECG changes:

- Shift potassium intracellularly with glucose/insulin: 10 units short-acting insulin and 25 g glucose (50 ml 50 % glucose or 125 ml 20 % glucose) IV over 15-30 min (onset in 15-30 min; maximal effect at 30-60 min; monitor blood glucose).

- Consider dialysis guided by clinical setting (immediate onset). Seek expert help.

Severe elevation (≥ 6.5 mmol l⁻¹) without ECG changes:

- Seek expert help.

- Glucose/insulin *(see above)*

- Salbutamol 10-20 mg nebulised (onset in 15-30 min; duration of action 4-6 h)

- Remove potassium from the body (consider dialysis)

There is insufficient evidence to support the use of sodium bicarbonate to decrease serum potassium.

Severe elevation (≥ 6.5 mmol l⁻¹) WITH toxic ECG changes (figure 12.1):

- Seek expert help.

- Protect the heart with calcium chloride: 10 ml 10 % calcium chloride IV or 30 mL 10 % calcium gluconate IV over 5-10 min to antagonise the toxic effects of hyperkalaemia at the myocardial cell membrane. This protects the heart by reducing the risk of VF/pVT, but does not lower serum potassium (onset in 1-3 min). Ensure secure vascular access prior to administration (risk of tissue necrosis secondary to extravasation of calcium salts).

- Use shifting strategies stated above (glucose/insulin and salbutamol).

- Remove potassium from the body (consider dialysis). Prompt specialist referral is essential. In hospitals without a dedicated renal unit, intensive care units can often provide emergency renal replacement therapies.

Patient in cardiac arrest

Modifications to BLS
There are no modifications to basic life support in the presence of electrolyte abnormalities.

Modifications to ALS
Follow the ALS algorithm. Confirm hyperkalaemia using a blood gas analyser if available.

Cardiac arrest: protect the heart first; then use shifting and removal strategies.

- Calcium chloride: 10 ml 10% calcium chloride IV by rapid bolus injection to antagonise the toxic effects of hyperkalaemia at the myocardial cell membrane.

- Glucose/insulin: 10 units short-acting insulin and 25 g glucose IV by rapid injection.

- Sodium bicarbonate: 50 mmol IV by rapid injection (if severe acidosis or renal failure).

- Consider dialysis for hyperkalaemic cardiac arrest resistant to medical treatment. Several dialysis modalities have been used safely and effectively in cardiac arrest, but this may only be available in specialist centres.

Indications for dialysis

Dialysis is the most effective method for removal of potassium from the body. The principle mechanism of action is the diffusion of potassium ions across the membrane down the potassium ion gradient. The typical decline in serum potassium is 1 mmol l^{-1} in the first 60 min, followed by 1 mmol l^{-1} over the next 2 h.

The main indications for dialysis in patients with hyperkalaemia are:
- severe life-threatening hyperkalaemia with or without ECG changes or arrhythmia

- hyperkalaemia resistant to medical treatment

- end-stage renal disease

- oliguric acute kidney injury (< 400 ml day^{-1} urine output)

- marked tissue breakdown (e.g. rhabdomyolysis)

Serum potassium frequently rebounds after initial treatment. In unstable patients continuous renal replacement therapy (e.g. continuous veno-veno haemodiafiltration) is less likely to compromise cardiac output than intermittent haemodialysis. This is now widely available in many intensive care units.

Cardiac arrest during haemodialysis

- Sudden cardiac death is the most common cause of death in haemodialysis patients and is usually preceded by ventricular arrhythmias. The frequency of cardiac arrest is highest on the first session of haemodialysis of the week (i.e. Monday or Tuesday) as fluid and electrolyte disturbances peak after the weekend interval.

- Call the resuscitation team and seek expert help immediately.

- Follow the universal ALS algorithm.

- Assign a trained dialysis nurse to operate the dialysis machine. Stop ultrafiltration (i.e. fluid removal) and give a fluid bolus. Return the patient's blood volume and disconnect from the dialysis machine.

- Leave dialysis access open and use for emergency drug administration in life-threatening circumstances and cardiac arrest.

- Minimise delay in delivering defibrillation. VF/pVT is more common in patients undergoing haemodialysis than in the general population. Most haemodialysis

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machine manufacturers recommend disconnection from dialysis equipment for defibrillation. Beware of wet surfaces (i.e dialysis machines may leak).

- Exclude all the reversible causes (4 Hs and 4 Ts). Electrolyte disorders, particularly hyperkalaemia, and fluid overload (e.g. pulmonary oedema) are most common causes of cardiac arrest.

2.2.2. Hypokalaemia

Hypokalaemia is the most common electrolyte disorder in clinical practice. It is seen in up to 20% of hospitalised patients. Hypokalaemia increases the incidence of arrhythmias, particularly in patients with pre-existing heart disease and in those treated with digoxin.

Definition

Hypokalaemia is defined as serum potassium < 3.5 mmol l^{-1}. Severe hypokalaemia is defined as potassium < 2.5 mmo L^{-1} and may be associated with symptoms.

Causes
Causes of hypokalaemia include:
- gastrointestinal losses (diarrhea)

- drugs (diuretics, laxatives, steroids)

- renal losses (renal tubular disorders, diabetes insipidus, dialysis)

- endocrine disorders (Cushing's syndrome, hyperaldosteronism)

- metabolic alkalosis

- magnesium depletion

- poor dietary intake

Treatment for hyperkalaemia can also induce hypokalaemia.

Recognition of hypokalaemia
Exclude hypokalaemia in every patient with an arrhythmia or cardiac arrest. In dialysis patients, hypokalaemia occurs commonly at the end of a haemodialysis session or during treatment with continuous ambulatory peritoneal dialysis (CAPD).

As serum potassium concentration decreases, the nerves and muscles are predominantly affected, causing fatigue, weakness, leg cramps or constipation. In severe cases (K^+< 2.5 mmol l^{-1}), rhabdomyolysis, ascending paralysis and respiratory difficulties may occur.

ECG features of hypokalaemia are:
- U waves

- T wave flattening

- ST segment changes

- arrhythmias (especially if patient is taking digoxin)

- cardiorespiratory arrest (PEA, VF/pVT, asystole)

Treatment of hypokalaemia

This depends on the severity of hypokalaemia and the presence of symptoms and ECG abnormalities. Gradual replacement of potassium is preferable, but in an emergency, intravenous potassium is required. Seek expert help. The maximum recommended IV infusion rate of potassium is 20 mmol h^{-1}, but more rapid infusion (e.g. 2 mmol min^{-1} for 10 min, followed by 10 mmol over 5-10 min) is indicated for unstable arrhythmias when cardiac arrest is imminent. Continuous ECG monitoring is essential during IV infusion. Adjust the dose after repeated sampling of serum potassium levels.

Patients who are potassium deficient can also be deficient in magnesium. Repletion of magnesium stores will facilitate more rapid correction of hypokalaemia and is recommended in severe cases of hypokalaemia.

2.2.3. Calcium and magnesium disorders

The recognition and management of calcium and magnesium disorders is summarised in table 12.1.

Table 12.1
Calcium and magnesium disorders

Disorder	Causes	Presentation	ECG	Treatment
Hypercalcaemia Total Calcium* > 2.6 mmol l^{-1}	Primary or tertiary hyperparathyroidism Malignancy Sarcoidosis Drugs	Confusion Weakness Abdominal pain Hypotension Arrhythmias Cardiac arrest	Short QT interval Prolonged QRS Interval Flat T waves AV-block	Fluid replacement IV Furosemide 1 mg kg^{-1} IV Hydrocortisone 200-300 mg IV Pamidronate 30-90 mg IV Treat underlying cause
Hypocalcaemia Total Calcium* < 2.1 mmol l^{-1}	Chronic renal failure Acute pancreatitis Calcium channel blocker overdose Toxic shock syndrome Rhabdomyolysis Tumour lysis syndrome	Paraesthesia Tetany Seizures AV-block Cardiac arrest	Prolonged QT interval T wave inversion Heart block	Calcium chloride 10 % 10-40 ml IV Magnesium sulphate 50 % 4-8 mmol (if necessary) IV

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Disorder	Causes	Presentation	ECG	Treatment
Hypermagnesaemia Magnesium > 1.1 mmol l^{-1}	Renal failure Iatrogenic	Confusion Weakness Respiratory depression AV-block Cardiac arrest	Prolonged PR and QT intervals T wave peaking AV-block	Consider treatment when magnesium > 1.75 mmol l^{-1} Calcium chloride 10 % 5-10 ml IV repeated if necessary Ventilatory support if necessary Saline diuresis: 0.9 % saline with furosemide 1 mg kg^{-1} IV Haemodialysis
Hypomagnesaemia Magnesium < 0.6 mmol l^{-1}	GI loss Polyuria Starvation Alcoholism Malabsorption	Tremor Ataxia Nystagmus Seizures Arrhythmias – torsade de pointes Cardiac arrest	Prolonged PR and QT Intervals ST-segment depression T-wave inversion Flattened P waves Increased QRS duration Torsade de pointes	Severe or symptomatic: 2 g 50 % magnesium sulphate (4 ml; 8 mmol) IV over 15 min Torsade de pointes: 2 g 50 % magnesium sulphate (4 ml; 8 mmol) IV over 1-2 min Seizure: 2 g 50 % magnesium sulphate (4 ml; 8 mmol) IV over 10 min

* A normal total calcium is about 2.2 to 2.6 mmol l^{1}. A *normal ionized calcium* is about 1.1 to 1.3 mmol l^{-1}. Calcium values need to be interpreted with caution. Seek expert help if not sure. Total calcium depends on serum albumin values and will need to be corrected for low albumin values (corrected total calcium). Ionized calcium values are often measured by blood gas machines. It is important not to confuse ionized calcium, total calcium and corrected calcium values.

3. Poisoning

Poisoning rarely causes cardiac arrest or death, but hospital admissions due to non-traumatic coma are common.

Poisoning by therapeutic or recreational drugs and by household products are the main reasons for hospital admission and poison centre calls. Inappropriate dosing and drug interactions can also cause drug toxicity. Accidental poisoning is commonest in children. Homicidal poisoning is uncommon.

Industrial accidents, warfare or terrorism can also cause exposure to toxins. Decontamination and safe management for individual or mass casualty incidents is not part of this manual.

3.1. Initial treatment

Supportive care based on the ABCDE (Airway, Breathing, Circulation, Disability, Exposure) approach with correction of hypoxia, hypotension, acid/base, and electrolyte disorders to prevent cardiorespiratory arrest whilst awaiting drug elimination is the mainstay of treatment.

- Airway obstruction and respiratory arrest secondary to a decreased conscious level is a common cause of death after self-poisoning (benzodiazepines, alcohol, opiates, tricyclics, barbiturates).

- Ensure your personal safety when there is a suspicious cause or unexpected cardiac arrest.

- Avoid mouth-to-mouth ventilation in the presence of toxins such as cyanide, hydrogen sulphide, corrosives and organophosphates. Ventilate the patient's lungs using a pocket mask or bag-mask and the highest possible concentration of oxygen.

- There is a high incidence of pulmonary aspiration of gastric contents after poisoning. In unconscious patients who cannot protect their airway, use a rapid sequence induction to intubate the trachea and decrease the risk of aspiration. This must be undertaken by persons trained and competent in the technique.

- Cardioversion is indicated for life-threatening tachyarrhythmia. Use the guidelines for peri-arrest arrhythmias *(chapter 11)*. Correct electrolyte and acid-base abnormalities.

- Drug-induced hypotension is common after self-poisoning. This usually responds to fluid therapy, but occasionally vasopressors (e.g. noradrenaline infusion) are required.

- Provide standard basic and advanced life support if cardiac arrest occurs.

- Once resuscitation is under way, try to identify the poison(s). Relatives, friends and ambulance crews can usually provide useful information. Patient examination may give diagnostic clues such as odours, needle puncture marks, pinpoint pupils, tablet residues, signs of corrosion in the mouth, or blisters associated with prolonged coma.

- Measure the patient's temperature - hypo- or hyperthermia may occur after drug overdose.

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- Consider prolonged resuscitation measures, particularly in young patients, as the poison may be metabolised or excreted.

- Consult a regional or national poisons centre for information on treatment of the poisoned patient. Specialist advice about specific poisons can be obtained by national databases. The World Health Organization lists poison centres at: *http://apps.who.int/poisoncentres.*

3.2. Specific treatments

There are few specific therapies for poisons that are useful immediately: decontamination, enhancing elimination, and the use of specific antidotes. Seek advice from a poisons centre for up-to-date guidance for severe or uncommon poisonings.

- Activated charcoal adsorbs most drugs. It is most effective if given within 1 h of the time of the ingestion. There is little evidence that treatment with activated charcoal improves clinical outcome. Consider giving a single dose of activated charcoal to patients who have ingested a potentially toxic amount of a poison. Give only to patients with an intact or protected airway. Multiple doses may be beneficial in life-threatening poisoning with carbemazepine, dapsone, phenobarbital, quinine and theophylline. Activated charcoal does not bind lithium, heavy metals and toxic alcohols.

- Routine use of gastric lavage for gastrointestinal decontamination is not recommended. In the rare instances (e.g. lethal ingestion with recent exposure), it should only be performed by individuals with proper training and expertise. It is contraindicated if the airway is not protected and if a hydrocarbon with high aspiration potential or a corrosive substance has been ingested.

- Whole-bowel irrigation can reduce drug absorption by cleansing the gastro-intestinal tract by enteral administration of a polyethylene glycol solution. Consider in potentially toxic ingestion of sustained release or enteric-coated drugs, oral iron or lithium poisoning, and the removal of ingested packets of illicit drugs.

- Avoid routine administration of laxatives (cathartics) and do not use emetics (e.g. ipecac syrup).

- Urinary alkalinisation (urine pH ≥ 7.5) by giving IV sodium bicarbonate can be useful in moderate to severe salicylate poisoning in patients who do not need haemodialysis).

- Haemodialysis removes drugs or metabolites with low molecular weight, low protein binding, small volumes of distribution and high water solubility. In case of hypotension, use continuous veno-venous hemofiltration (CVVH) or continuous veno-venous haemodialysis (CVVHD) alternatively. Seek expert help and/or consult a poisons centre for information on treatment.

- Consider the use of lipid emulsion (Intralipid) for cardiac arrest caused by local anaesthetic toxicity *(see below)*.

- Specific antidotes include: acetylcysteine for paracetamol; atropine for organophosphate insecticides; sodium nitrite, sodium thiosulfate, hydroxocobalamin, and amyl nitrite for cyanides; digoxin-specific Fab antibodies for

digoxin; flumazenil for benzodiazepines; naloxone for opioids; calcium chloride for calcium channel blockers.

3.3. Specific poisonings

This section addresses only some causes of cardiac arrest from poisoning.

3.3.1. Opioid poisoning

Opioid poisoning causes respiratory depression, pinpoint pupils and coma followed by respiratory arrest. The opioid antagonist naloxone rapidly reverses these effects. There are fewer adverse events when the airway is opened and patients receive oxygen and ventilation (e.g. with pocket mask or bag-mask) before naloxone in opioid-induced respiratory depression; however, the use of naloxone may prevent the need for intubation.

The route for giving naloxone depends on the skills of the rescuer: intravenous (IV), intramuscular (IM), subcutaneous (SC), and intranasal (IN) routes can be used. Sometimes combinations are helpful. The non-IV routes may be quicker because time is saved in not having to establish IV access, which can be extremely difficult in an IV drug abuser. Additionally naloxone is released slower using the non-IV routes thus increasing the duration of action. The initial doses of naloxone are 0.4-2 mg IV, IO, IM or SC, and may be repeated every 2-3 min. Additional doses may be needed every 20-60 min. Intranasal dosing is 2 mg IN (1 mg in each nostril) which may be repeated every 5 min. Titrate the dose until the victim is breathing adequately and has protective airway reflexes. Large opioid overdoses may require a total dose of up to 10 mg of naloxone. All patients treated with naloxone must be monitored. The duration of action of naloxone is 45-70 min, but respiratory depression may persist for 4-5 h after opioid overdose.

Acute withdrawal from opioids produces a state of sympathetic excess and can cause complications such as pulmonary oedema, ventricular arrhythmia, and severe agitation. Use naloxone reversal of opioid intoxication with caution in patients suspected of opioid dependence.

Cardiac arrest is usually secondary to a respiratory arrest and associated with severe brain hypoxia. Prognosis is poor. There are no data on the use of any additional therapies beyond standard ALS guidelines in opioid-induced cardiac arrest.

3.3.2. Benzodiazepines

Overdose of benzodiazepines can cause loss of consciousness, respiratory depression and hypotension. Flumazenil, a competitive antagonist of benzodiazepines, should be used for reversal of sedation caused by a single ingestion of any of the benzodiazepines and when there is no history or risk of seizures. Reversal of benzodiazepine intoxication with flumazenil can cause significant toxicity (seizure, arrhythmia, hypotension, and withdrawal syndrome) in patients with benzodiazepine dependence or co-ingestion of proconvulsant medications such as tricyclic antidepressants. Do not use flumazenil routinely in the comatose overdose patient. There are no specific modifications required for cardiac arrest caused by benzodiazepines.

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3.3.3. Tricyclic antidepressants

This includes tricyclic and related cyclic drugs (e.g. amitriptyline, desipramine, imipramine, nortriptyline, doxepin, and clomipramine). Self-poisoning with tricyclic antidepressants is common and can cause hypotension, seizures, coma and life-threatening arrhythmias. Cardiac toxicity mediated by anticholinergic and sodium channel-blocking effects can produce a broad-complex tachycardia (VT). Hypotension is exacerbated by alpha-1 receptor blockade. Anticholinergic effects include mydriasis, fever, dry skin, delirium, tachycardia, ileus, and urinary retention. Most life-threatening problems occur within the first 6 h after ingestion.

A widening QRS complex and right axis deviation indicates a greater risk of arrhythmias *(figure 12.3)*. Give sodium bicarbonate (1-2 mmol kg^{-1}) for the treatment of tricyclic-induced ventricular arrhythmias. While no study has investigated the optimal target arterial pH with bicarbonate therapy, a pH of 7.45-7.55 has been commonly accepted.

3.3.4. Local anaesthetic toxicity

Local anaesthetic toxicity occurs typically in the setting of regional anaesthesia, when a bolus of local anaesthetic inadvertently enters an artery or vein. Systemic toxicity of local anaesthetics involves the central nervous system, and the cardiovascular system. Severe agitation, loss of consciousness, with or without tonic-clonic convulsions, sinus bradycardia, conduction blocks, asystole and ventricular tachyarrhythmia can all occur. Toxicity can be potentiated in pregnancy, extremes of age, or hypoxaemia. In cases of cardiac arrest prolonged times of CPR may be necessary to achieve ROSC.

Patients with both cardiovascular collapse and cardiac arrest attributable to local anaesthetic toxicity may benefit from treatment with intravenous 20% lipid emulsion in addition to standard advanced life support. Give an initial intravenous bolus injection of 20% lipid emulsion 1.5 ml kg^{-1} over 1 min followed by an infusion at 15 ml kg^{-1} h^{-1}. Give up to a maximum of two repeat boluses at 5-min intervals and continue until the patient is stable. The maximum cumulative dose within the first 30 minutes should not extend 12 ml kg^{-1} of lipid emulsion.

3.3.5. Calcium channel blockers

Calcium channel blocker overdose is emerging as a common cause of prescription drug poisoning deaths. The treatment for calcium channel blocker poisoning is supported by low-quality evidence.

Give calcium chloride 10% in boluses of 20 ml (or equivalent dose of calcium gluconate) every 2-5 min in severe bradycardia or hypotension followed by an infusion if needed. While calcium in high doses can overcome some of the adverse effects, it rarely restores normal cardiovascular status. Haemodynamic instability may respond to high doses of insulin (1 unit kg^{-1} followed by an infusion of 0.5-2.0 units kg^{-1} h^{-1}) given with glucose supplementation and electrolyte monitoring in addition to standard treatments including fluids and vasopressors.

3.3.6. Beta-blockers

Beta-blocker toxicity causes bradyarrhythmias and negative inotropic effects that are difficult to treat, and can lead to cardiac arrest.

Evidence for treatment is based on case reports and animal studies. Improvement has been reported with glucagon (50-150 mcg kg^{-1}), high-dose insulin and glucose, lipid emulsions, phosphodiesterase inhibitors, extracorporeal and intra-aortic balloon pump support, and calcium salts.

3.3.7. Digoxin

Although cases of digoxin poisoning are fewer than those involving calcium channel and beta-blockers, the mortality rate from digoxin is far greater. Specific antidote therapy with digoxin-specific antibody fragments (digoxin-Fab) should be used. Give 2-10 vials digoxin-Fab (38 mg per vial) IV over 30 min.

Figure 12.3
12-lead ECG showing features of severe tricyclic antidepressant toxicity

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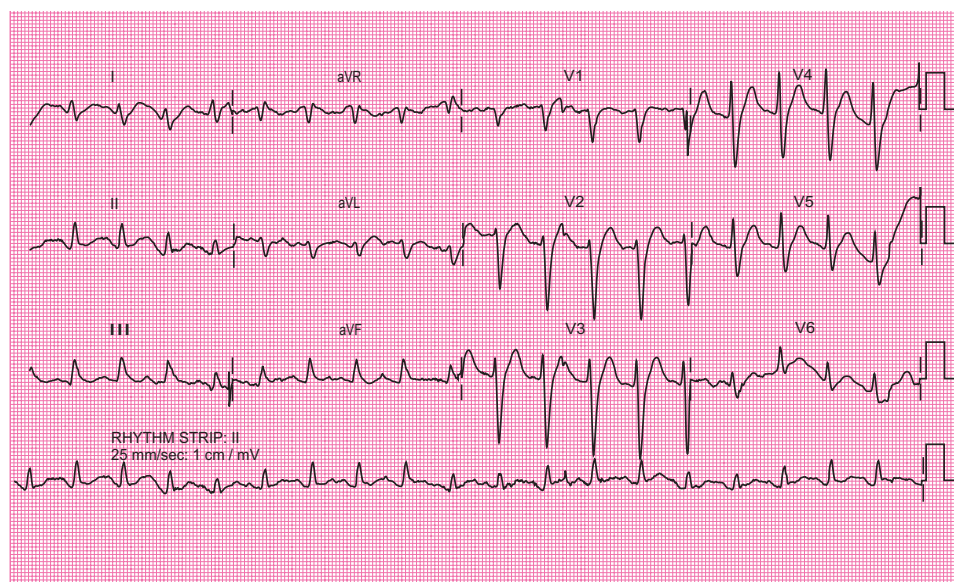
3.4. Further treatment and prognosis

A long period of coma in a single position can cause pressure sores and rhabdomyolysis. Measure electrolytes (particularly potassium), blood glucose and arterial blood gas values. Monitor temperature because thermoregulation is impaired. Both hypothermia and hyperthermia (hyperpyrexia) can occur after overdose of some drugs. Retain samples of

blood and urine for analysis. Be prepared to continue resuscitation for a prolonged period, particularly in young patients, as the poison may be metabolised or excreted during extended life support measures.

4. Hypothermia

4.1. Definition

Hypothermia exists when the body core temperature is below 35° C and is classified arbitrarily as mild (32-35° C), moderate (28-32° C), or severe (< 28° C). The Swiss staging system based on clinical signs can be used by rescuers at the scene to describe victims: stage I - conscious and shivering; stage II - impaired consciousness without shivering; stage III - unconscious; stage IV – cardiac arrest or low flow state and V - death due to irreversible hypothermia.

4.2. Diagnosis

Accidental hypothermia may be under-diagnosed in countries with a temperate climate. In people with normal thermoregulation, hypothermia can develop during exposure to cold environments, particularly wet or windy conditions, and in people who have been immobilised, or following immersion in cold water. When thermoregulation is impaired, for example, in the elderly and very young, hypothermia can follow a mild insult. The risk of hypothermia is also increased by drug or alcohol ingestion, exhaustion, illness, injury or neglect especially when there is a decrease in the level of consciousness. Hypothermia may be suspected from the clinical history or a brief external examination of a collapsed patient. A low-reading thermometer is needed to measure the core temperature and confirm the diagnosis. The core temperature measured in the lower third of the oesophagus correlates well with the temperature of the heart. 'Tympanic' measurement - using a thermistor technique - is a reliable alternative but may be lower than the oesophageal temperature if the environmental temperature is very cold, the probe is not well insulated, the external auditory canal is blocked or during cardiac arrest when there is no flow in the carotid artery. Widely available 'tympanic' thermometers based on infrared technique do not seal the ear canal and are often not suitable for low temperature readings.

4.3. Decision to resuscitate

Cooling of the human body decreases cellular oxygen consumption by about 6 % per 1°C decrease in core temperature. In some cases, hypothermia can exert a protective effect on the brain and vital organs and intact neurological recovery is possible even after prolonged cardiac arrest if deep hypothermia develops before asphyxia.

Beware of diagnosing death in a hypothermic patient because cold alone may produce a very slow, small-volume, irregular pulse and unrecordable blood pressure. In a hypothermic patient, no signs of life (Swiss hypothermia stage IV) alone are unreliable for declaring death. At 18° C the brain can tolerate periods of circulatory arrest for ten times longer than at 37° C. Dilated pupils can be caused by a variety of insults and must not be regarded as

a sign of death. Good quality survival has been reported after cardiac arrest and a core temperature of 13.7° C after immersion in cold water with prolonged CPR.

In the pre-hospital setting, resuscitation should be withheld only if the cause of a cardiac arrest is clearly attributable to a lethal injury, fatal illness, prolonged asphyxia, or if the chest is incompressible. In all other patients the traditional guiding principle that "no one is dead until warm and dead" should be considered. In remote wilderness areas, the impracticalities of achieving rewarming have to be considered. In the hospital setting involve senior doctors and use clinical judgment to determine when to stop resuscitating a hypothermic arrest victim.

4.4. Treatment of hypothermia

The standard principles of prevention and life support apply to the hypothermic patient. Do not delay urgent procedures, such as tracheal intubation and insertion of vascular catheters.

- Open the airway and, if there is no spontaneous respiratory effort, ventilate the patient's lungs with high concentrations of oxygen. If possible, use warmed (40-46°C) and humidified oxygen. Consider careful tracheal intubation when indicated according to the ALS algorithm. Procedures can precipitate VF. However, the advantages of adequate oxygenation and protection from aspiration outweigh the minimal risk of triggering VF by performing tracheal intubation.

- Palpate a major artery and, if available, look at the ECG for up to 1 min and look for signs of life before concluding that there is no cardiac output. Both the respiratory rate and pulse may be very slow in severe hypothermia so more assessment time is necessary. Echocardiography or Doppler ultrasound can be used to establish if there is a cardiac output or peripheral blood flow.

- If the victim is pulseless, start chest compressions immediately. Use the same ventilation and chest compression rates as for a normothermic patient. Hypothermia can cause stiffness of the chest wall, making ventilation and chest compressions more difficult. If you are not experienced in patient assessment or if there is any doubt about whether a pulse is present, start chest compressions until more experienced help is available.

- Once resuscitation is underway, confirm hypothermia with a low reading thermometer. Use oesophageal, bladder, rectal, or tympanic temperature measurements. Try to use a consistent method to allow serial comparisons of temperature.

- The hypothermic heart may be unresponsive to cardio-active drugs, attempted electrical pacing, and attempted defibrillation. Drug metabolism is slowed, leading to potentially toxic plasma concentrations of any drugs given repeatedly. Withhold adrenaline and other drugs until the patient has been warmed to a temperature greater than about 30°C. Once 30°C has been reached, double the intervals between doses (twice as long as normal). As the patient's temperature returns towards normal (above 35° C), use the standard drug protocols.

- Give drugs via a central or large proximal vein if possible.

- Remember to rule out other primary causes of cardiorespiratory arrest (e.g. drug overdose, hypothyroidism or trauma) or reversible causes using the four Hs and four Ts approach.

- Monitor electrolytes, glucose and blood gases regularly during resuscitation and post-resuscitation care as rapid changes can occur.

- Blood gas analysers will give blood gas values for a temperature of 37° C unless the patient's temperature is entered in to the analyser. Oxygen and carbon dioxide partial pressures are lower in hypothermia because gases become more soluble as blood temperature decreases. In clinical practice it is much easier to make all the measurements at 37° C i.e. temperature uncorrected values. It is then only necessary to compare them with the well-known normal values for 37° C. This also enables comparison of serial results from blood gas samples taken during rewarming.

4.4.1. Arrhythmias

As the body core temperature decreases, sinus bradycardia tends to give way to atrial fibrillation (AF) followed by ventricular fibrillation (VF), and finally asystole.

- If VF/pVT is detected, give initial shocks according to standard ALS treatment protocols; if VF/pVT persists after three shocks, delay further defibrillation attempts until the core temperature is above 30° C. If an AED is used, follow the AED prompts while rewarming the patient.

- Arrhythmias other than VF tend to revert spontaneously as the core temperature increases and usually do not require immediate treatment.

- Bradycardia can be physiological in severe hypothermia. Cardiac pacing is not indicated unless the bradycardia persists after rewarming.

4.4.2. Rewarming

General measures for all victims include removal from the cold environment, prevention of further heat loss and rapid transfer to the most appropriate hospital. Rewarming may be passive, active external, or active internal.

- In the field, a patient with moderate or severe hypothermia should be immobilised and handled carefully, oxygenated adequately, monitored (including ECG and core temperature), and the whole body dried and insulated. Wet clothes should be cut off rather than stripped off; this will avoid excessive movement of the victim.

- Conscious victims can mobilise as exercise re-warms a person more rapidly than shivering. Exercise can increase any after-drop, i.e. further cooling after removal from a cold environment. Somnolent or comatose victims have a low threshold for developing VF or pVT and should be immobilised and kept horizontal to avoid an after-drop or cardiovascular collapse.

- Passive rewarming is appropriate in conscious victims with mild hypothermia

who are still able to shiver. This is best achieved by full body insulation with wool blankets, aluminium foil, a hat and warm environment.

- The application of chemical heat packs to the trunk is particularly helpful in moderate and severe hypothermia to prevent further heat loss in the pre-hospital setting.

- Rewarming in the field with heated intravenous fluids and warm humidified gases is not efficient. Intensive active rewarming must not delay transport to a hospital where advanced rewarming techniques, continuous monitoring and observation are available.

In general, alert hypothermic and shivering victims without an arrhythmia can be transported to the nearest hospital for passive rewarming and observation. Hypothermic victims with an altered consciousness should be taken to a hospital capable of active external and internal rewarming. If any signs of cardiac instability are present, transport the patient to an extracorporeal life support (ECLS) centre, contacting them well in advance to ensure that the hospital can accept the patient for extracorporeal rewarming.

- Active external rewarming techniques include forced air rewarming and warmed (up to 42°C) intravenous fluids. These techniques are effective (rewarming rate 1-1.5°C h^{-1}) in patients with severe hypothermia and a perfusing rhythm.

- Active internal rewarming techniques include warm humidified gases; gastric, peritoneal, pleural or bladder lavage with warmed fluids (at 40°C), and extracorporeal rewarming.

- In a hypothermic patient with apnoea and cardiac arrest, extracorporeal rewarming is the preferred method of active internal rewarming because it provides sufficient circulation and oxygenation while the core body temperature is increased by 8-12°C h^{-1}. Survivors in one case series had an average of 65 min of conventional CPR before cardiopulmonary bypass. Unfortunately, facilities for extracorporeal rewarming are not always available and a combination of rewarming techniques may have to be used.

- During rewarming, patients will require large volumes of fluids as vasodilation causes expansion of the intravascular space. Continuous haemodynamic monitoring and warm IV fluids are essential.

- Avoid hyperthermia during and after rewarming. Once ROSC has been achieved use standard post-resuscitation care.

4.5. Avalanche burial

In Europe and North America, there are about 150 snow avalanche deaths each year. Most are sports-related and involve skiers, snowboarders and snowmobilers. Death from avalanches is due to asphyxia, trauma or hypothermia. Avalanches occur in areas that are difficult to access by rescuers in a timely manner, and burials frequently involve multiple victims.

Avalanche victims are not likely to survive when they are:
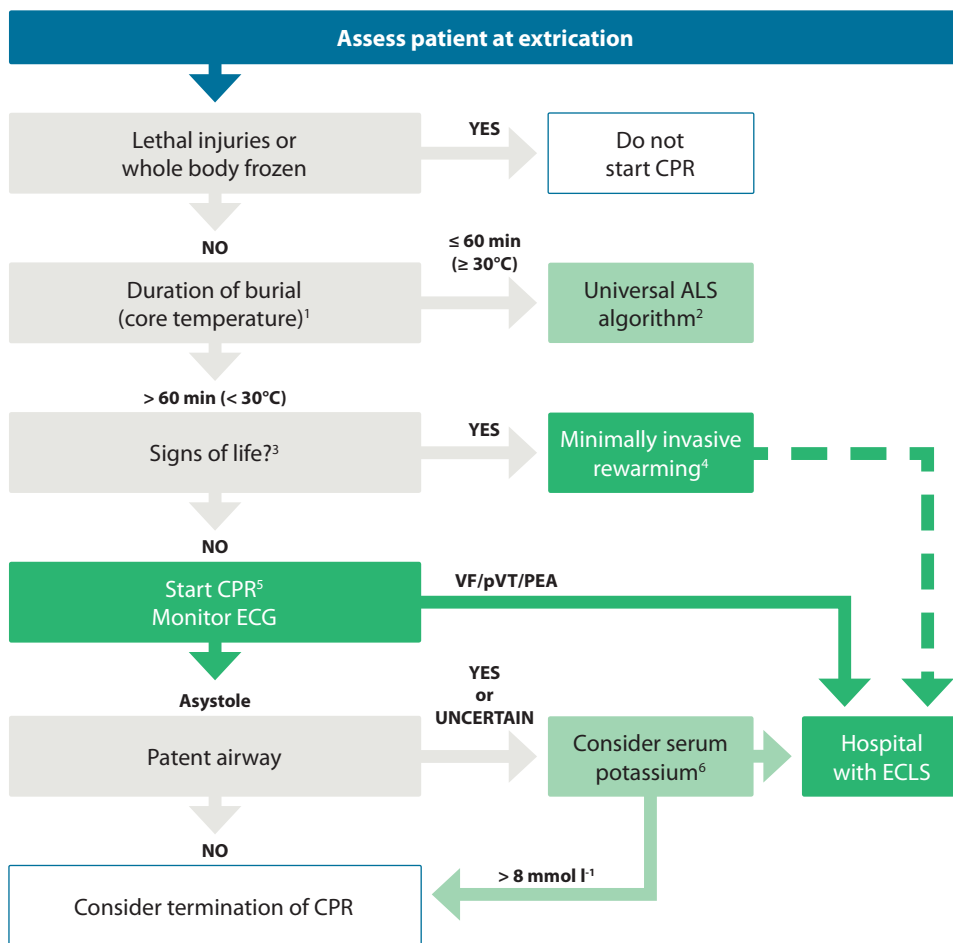
- buried > 60 min (or if the initial core temperature is < 30°C) and in cardiac arrest with an obstructed airway on extrication;

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- buried and in cardiac arrest on extrication with an initial serum potassium > 8 mmol l^{-1}.

The algorithm for the management of buried avalanche victims is shown in figure 12.4.

Figure 12.4
Avalanche accident algorithm for completely buried victims.

5. Hyperthermia

5.1. Definition

Hyperthermia occurs when the body's ability to thermoregulate fails and core temperature exceeds that normally maintained by homeostatic mechanisms. Hyperthermia may be exogenous, caused by environmental conditions or secondary to endogenous heat production.

Environment-related hyperthermia occurs where heat, usually in the form of radiant energy, is absorbed by the body at a rate faster than can be lost by thermoregulatory mechanisms. Hyperthermia occurs along a continuum of heat-related conditions starting with heat stress, progressing to heat exhaustion, heat stroke and culminating in multi-organ dysfunction and cardiac arrest in some instances.

Malignant hyperthermia (MH) is a rare disorder of skeletal muscle calcium homeostasis characterised by muscle contracture and life-threatening hypermetabolic crisis following exposure of genetically predisposed individuals to halogenated anaesthetics and depolarising muscle relaxants.

5.2. Heat stroke

Heat stroke is a systemic inflammatory response with a core temperature above 40°C (104°F) accompanied by mental state change and varying levels of organ dysfunction. There are two forms of heat stroke: classic non-exertional heat stroke occurs during high environmental temperatures and often affects the elderly during heat waves; exertional heat stroke occurs during strenuous physical exercise in high environmental temperatures and/or high humidity and usually effects healthy young adults. Mortality from heat stroke ranges between 10-50 %.

5.2.1. Predisposing factors

The elderly are at increased risk for heat-related illness because of underlying illness, medication use, declining thermoregulatory mechanisms, and limited social support. There are several risk factors: lack of acclimatisation, dehydration, obesity, alcohol, cardiovascular disease, skin conditions (psoriasis, eczema, scleroderma, burn, cystic fibrosis), hyperthyroidism, phaeochromocytoma, and drugs (anticholinergics, diamorphine, cocaine, amphetamine, phenothiazines, sympathomimetics, calcium channel blockers, beta blockers).

5.2.2. Clinical presentation

Heat stroke can resemble septic shock and may be caused by similar mechanisms. Features include:

- core temperature 40°C or more
- hot, dry skin (sweating is present in half cases of exertional heat stroke)

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- early signs and symptoms include: extreme fatigue, headache, fainting, facial flushing, vomiting and diarrhoea

- cardiovascular dysfunction including arrhythmias and hypotension

- respiratory dysfunction including ARDS

- central nervous system dysfunction including seizures and coma

- liver and renal failure

- coagulopathy

- rhabdomyolysis

Other clinical conditions need to be considered, including:

- drug toxicity

- drug withdrawal syndrome

- serotonin syndrome

- neuroleptic malignant syndrome

- sepsis

- central nervous system infection

- endocrine disorders (e.g. thyroid storm, phaeochromocytoma)

5.2.3. Treatment

The mainstay of treatment is supportive therapy based on optimising the ABCDEs and rapidly cooling the patient.

- Start cooling before the patient reaches hospital. Aim to rapidly reduce the core temperature to approximately 39°C. Patients with severe heat stroke need to be managed in a critical care setting.

- Use haemodynamic monitoring to guide fluid therapy. Large volumes of fluid may be required. Correct electrolyte abnormalities.

- If cardiac arrest occurs, follow standard procedures for basic and advanced life support and cool the patient. Attempt defibrillation, if appropriate, while continuing to cool the patient. Animal studies suggest the prognosis is poor compared with normothermic cardiac arrest. The risk of unfavourable neurological outcome increases for each degree of body temperature > 37°C.

- Provide standard post-resuscitation care *(chapter 13)*.

5.2.4. Cooling techniques

Several cooling methods have been described but there are few formal trials on which method is best.

- Simple techniques include drinking cold drinks, fanning the undressed patient and spraying tepid water on the patient. Ice packs over areas where there are large superficial blood vessels (axillae, groins, neck) are also useful. Surface cooling may cause shivering.

- In cooperative stable patients immersion in cold water is effective; however, this can cause peripheral vasoconstriction and reduce heat dissipation. Immersion is not practical in the sickest patients.

- Use the same advanced cooling techniques as used for targeted temperature management after cardiac arrest *(chapter 13)*. Consider the use of cold IV fluids, intravascular cooling catheters, surface cooling devices and extra corporeal circuits, e.g. continuous veno-veno haemofiltration or cardiopulmonary bypass.

- No specific drugs lower core temperature in heat stroke. There is no good evidence that antipyretics (e.g. non-steroidal anti-inflammatory drugs or paracetamol) are effective in heat stroke. Diazepam may be useful to treat seizures and facilitate cooling. Dantrolene *(see below)* has not been shown to be benefical.

5.3. Malignant hyperthermia

Malignant hyperthermia is a life-threatening genetic sensitivity of skeletal muscles to volatile anaesthetics and depolarising neuromuscular blocking drugs occurring during or after anaesthesia. Stop triggering agents immediately; give oxygen, correct acidosis and electrolyte abnormalities. Start active cooling and give dantrolene. Other drugs such as 3,4-methylenedioxymethamphetamine (MDMA, 'ecstasy') and amphetamines also cause a condition similar to malignant hyperthermia and the use of dantrolene may be benefical.

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6. Drowning

Drowning is a common cause of accidental death. The most important detrimental consequence of drowning is hypoxia. Cardiac arrest is usually a secondary event. The duration of hypoxia is a critical factor in determining the victim's outcome. Submersion durations of less than ten minutes are associated with a very high chance of favourable outcome, while submersion durations longer than 25 minutes are associated with a low chance of survival. Age, emergency medical services (EMS) response time, fresh or salt water, water temperature, and witness status are not useful predictors of survival.

Remember, some patients may have had a primary cardiac arrest (e.g. caused by myocardial infarction whilst swimming). Death from drowning is more common in young males, and is the leading cause of accidental death in Europe in this group.

6.1. Definition

Drowning is defined as a process resulting in primary respiratory impairment from submersion/immersion in a liquid medium. Implicit in this definition is that a liquid/air interface is present at the entrance of the victim's airway, preventing the victim from breathing air. The victim may live or die after this process, but whatever the outcome, he or she has been involved in a drowning incident.

Submersion occurs when the face is underwater or covered in water. Asphyxia and cardiac arrest occurs within a matter of minutes of submersion. Immersion, by contrast, is when the head remains above water, in most cases by means of the support of a lifejacket. In most situations of immersion, the victim remains immersed with an open airway and becomes hypothermic, although aspiration of water may occur if water splashes over the face or if the victim becomes unconscious with their face in the water.

6.2. Decision to resuscitate

Deciding whether to start or stop resuscitation of a drowning victim is notoriously difficult. No single factor predicts prognosis accurately.

- Start and continue resuscitation unless there is clear evidence that resuscitation attempts are futile (e.g. massive traumatic injuries, rigor mortis, putrefaction etc.), or timely evacuation to a medical facility is not possible.

Neurologically intact survival has been reported in several victims submerged for longer than 25 minutes, however these rare case reports almost invariably occur in children submerged in ice-cold water, when immersion hypothermia has preceeded hypoxia or in submersion of car occupants.

6.3. Treatment

Treatment of a drowning victim involves four phases. These comprise:

1. water rescue

2. basic life support

3. advanced life support

4. post-resuscitation care.

6.3.1. Water rescue and basic life support

- Ensure personal safety and minimise the danger to yourself at all times. If possible, attempt to save the drowning victim without entering the water. Talk to the victim, use a rescue aid (e.g. stick or clothing), or throw a rope or buoyant rescue aid if the victim is close to dry land. Alternatively, use a boat or other water vehicle to help with the rescue. Avoid entry into the water whenever possible. If entry into the water is essential, take a buoyant rescue aid or flotation device. It is safer to enter the water with two rescuers than alone.

- Remove the victim from the water promptly. The chances of a drowning victim sustaining a spinal injury are very low. Spinal precautions are unnecessary unless there is a history of diving in shallow water, or signs of severe injury after water-slide use, waterskiing, kite-surfing, or watercraft racing. If the victim is pulseless and apnoeic, remove them from the water as quickly as possible while attempting to limit neck flexion and extension.

- Hypovolaemia after prolonged immersion may cause a circum-rescue collapse/arrest. Keep the victim in a horizontal position during and after retrieval from the water.

Ventilation

- The BLS sequence in drowning reflects the critical importance of rapid alleviation of hypoxia. Prompt initiation of rescue breathing or positive pressure ventilation increases survival.

- Give five initial ventilations as soon as possible. Inflation should take about 1 second and be sufficient to see the chest rise. If possible supplement ventilation with oxygen.

- Cricoid pressure applied by trained and skilled personel in casualties without a secured airway may reduce gastric inflation and enhance ventilation in drowning.

- Trained individuals may undertake in water ventilation ideally with the support of a buoyant rescue aid. If a rescuer, in general a surf-lifeguard, finds a non-responding drowning victim in deep open water, the rescuer may start ventilation when trained to do so before moving the victim to dry land or rescue craft.

Chest compression

- As soon as the victim is removed from the water, check for breathing. If the victim is not breathing (or is making occasional gasps) after the initial ventilations, start chest compressions immediately. Continue CPR in a ratio of 30 compressions to 2 ventilations. Most drowning victims will have sustained cardiac arrest secondary to hypoxia. In these patients, compression-only CPR is likely to be ineffective and should be avoided.

Defibrillation

- Dry the victims chest before placing defibrillation electrodes. Standard procedures for defibrillation using an AED or manual defibrillator should be followed.

Fluid in the airway

- In some situations, massive amounts of foam caused by admixing moving air with water are seen coming out of the mouth of the victim. Do not try and attempt to remove the foam as it will keep coming. Continue rescue breaths/ventilation until an ALS provider arrives and is able to intubate the victim.

- Regurgitation of stomach contents and swallowed water is common during resuscitation from drowning. There is no need to clear the airway of aspirated water, which is absorbed rapidly into the central circulation. If this prevents ventilation completely, turn the victim on their side and remove the regurgitated material using directed suction if possible. If spinal cord injury is suspected, log-roll the victim, keeping the head, neck, and torso aligned. Log rolling requires several rescuers.

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6.3.2. Advanced life support

Airway and breathing

- During the initial assessment of the spontaneously breathing drowning victim, give high-flow oxygen (10-15 l min^{-1}), ideally through an oxygen mask with reservoir bag.

- Consider early tracheal intubation and controlled ventilation by skilled personnel for victims who fail to respond to these initial measures, who have a reduced level of consciousness or are in cardiac arrest. Reduced pulmonary compliance requiring high inflation pressures may limit the use of a supraglottic airway device. Take care to ensure optimal preoxygenation before attempting tracheal intubation. Pulmonary oedema fluid may pour from the airway and may need continuous suctioning to enable a view of the larynx. Confirm position of the tracheal tube.

- Titrate the inspired oxygen concentration to achieve a SpO$_2$ of 94-98%. Pulse oximetry can give spurious readings following rescue from drowning. Confirm adequate oxygenation and ventilation with arterial blood gases once available.

- Set positive end expiratory pressure (PEEP) to at least 10 cmH$_2$O. PEEP levels of 15-20 cmH$_2$O may be required if the patient is severely hypoxaemic.

- Decompress the stomach with a gastric tube.

Circulation and defibrillation

- Differentiating respiratory from cardiac arrest is particularly important in the drowning victim. Delaying the initiation of chest compressions if the victim is in cardiac arrest will reduce survival.

- The typical post-arrest gasping is very difficult to distinguish from the initial respiratory efforts of a spontaneous recovering drowning victim. Palpation of the pulse as the sole indicator of the presence or absence of cardiac arrest is unreliable. When available additional diagnostic information should be obtained from other monitoring modalities such as ECG trace, ETCO$_2$, echocardiography to confirm the diagnosis of cardiac arrest.

- If the victim is in cardiac arrest, follow standard advanced life support protocols. If the victims core body temperature is less than 30°C, limit defibrillation attempts to three, and withhold IV drugs until the core body temperature increases above 30°C.

- After prolonged immersion, most victims will have become hypovolaemic due to the cessation of the hydrostatic pressure of water on the body. Give rapid IV fluid to correct hypovolaemia. This should commence out-of-hospital if transfer time is prolonged.

6.3.3. Post-resuscitation care

- Victims of drowning are at risk of developing acute respiratory distress syndrome (ARDS) after submersion and protective ventilation strategies should be used.

- Pneumonia is common after drowning. Prophylactic antibiotics have not been shown to be of benefit, although they may be considered after submersion in

grossly contaminated water such as sewage. Give broad-spectrum antibiotics if signs of infection develop subsequently.

• There are no differences in the treatment of victims of fresh or sea water drowning.

• Attempts have been made to improve neurological outcome following drowning with the use of barbiturates, intracranial pressure (ICP) monitoring, and steroids. None of these interventions has altered outcome.

• Cardiac arrhythmias may cause rapid loss of consciousness leading to drowning if the victim is in water at the time. Take a careful history in survivors of a drowning incident to identify features suggestive of arrhythmic syncope. Symptoms may include syncope (whilst supine position, during exercise, with brief prodromal symptoms, repetitive episodes or associated with palpitations), seizures or a family history of sudden death. The absence of structural heart disease at post mortem does not rule the possibility of sudden cardiac death. Post mortem genetic analysis has proved helpful in these situations and should be considered if there is uncertainty over the cause of a drowning death.

7. Asthma

Worldwide, approximately 300 million people of all ages and ethnic backgrounds have asthma with a high prevalence in some European countries (United Kingdom, Ireland and Scandinavia). Annual worldwide deaths from asthma have been estimated at 250,000. The death rate does not appear to be correlated with asthma prevalence. Most deaths occur before hospital admission.

This guidance focuses on the treatment of patients with near-fatal asthma and cardiac arrest.

7.1. Patients at risk of asthma-related cardiac arrest

The risk of near-fatal asthma attacks is not necessarily related to asthma severity.

Patients most at risk include those with:

• a history of near-fatal asthma requiring intubation and mechanical ventilation

• a hospitalisation or emergency care for asthma in the past year

• low or no use of inhaled corticosteroids

• an increasing use and dependence of beta-2 agonists

• anxiety, depressive disorders and/or poor compliance with therapy

• food allergy in a patient with asthma

7.2. Causes of cardiac arrest

Cardiac arrest in the asthmatic is often a terminal event after a period of hypoxaemia; occasionally, it may be sudden. Cardiac arrest in asthmatics has been linked to:

- Severe bronchospasm and mucous plugging leading to asphyxia (most frequent cause of death).

- Cardiac arrhythmias due to hypoxia, stimulant drugs (e.g. ß-adrenergic agonists, aminophylline) or electrolyte abnormalities.

- Dynamic hyperinflation, i.e. auto-positive end-expiratory pressure (auto-PEEP), can occur in mechanically ventilated asthmatics. Auto-PEEP is caused by air trapping and 'breath stacking' (air entering the lungs and being unable to escape). Gradual build-up of pressure occurs and reduces venous return and blood pressure.

- Tension pneumothorax (often bilateral).

The 4 Hs and 4 Ts approach to reversible causes will help identify these causes in cardiac arrest.

7.3. Assessment and treatment

Use the ABCDE approach to assess severity and guide treatment. The severity of acute asthma is summarised in table 12.2.

Table 12.2
Severity of acute asthma exacerbations

Asthma severity		
Near-fatal asthma	Raised $PaCO_2$ and/or requiring mechanical ventilation with raised inflation pressures	
Life-threatening asthma	Any one of the following signs:	
	Clinical signs	**Measurements**
	Altered conscious level	PEF < 33 % best or predicted
	Exhaustion	SpO_2 < 92 %
	Arrhythmia	PaO_2 < 8 kPa
	Hypotension	'normal' $PaCO_2$ (4.6-6.0 kPa)
	Cyanosis	
	Silent chest	
	Poor expiratory effort	
Acute severe asthma	Any one of: - PEF 33-50 % best or predicted - respiratory rate ≥ 25 min^{-1} - heart rate ≥ 110 min^{-1} - inability to complete sentences in one breath	

PEF = peak expiratory flow

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- Wheezing is a common physical finding, but severity does not correlate with the degree of airway obstruction. Other causes of wheezing include: pulmonary oedema, chronic obstructive pulmonary disease (COPD), pneumonia, anaphylaxia, foreign bodies, pulmonary embolism, subglottic mass.

- The patient with acute severe asthma requires aggressive medical management to prevent deterioration. Experienced clinicians should treat these patients in a critical care area. Patients with SpO_2 < 92 % or with features of life-threatening asthma are at risk of hypercapnia and require arterial blood gas measurement.

- Use a concentration of inspired oxygen that will achieve an SpO_2 94-98 %. High-flow oxygen by mask is sometimes necessary. Lack of pulse oximetry should not prevent the use of oxygen.

- Salbutamol (5 mg nebulised) is the main therapy for acute asthma. Repeated doses every 15-20 min, or continuous doses, may be needed. Nebuliser units that can

be driven by high-flow oxygen (at least 6 l min^{-1}) should be used. Remember that nebulised drugs will not be delivered to the lungs effectively if the patient is tired and hypoventilating. If a nebuliser is not immediately available beta-2 agonists can be temporarily administered by repeating activations of a metered dose inhaler via a large volume spacer device.

- Nebulised anticholinergics (ipratropium 0.5 mg 4-6 hourly) may produce additional bronchodilation in severe asthma and in those who do not respond to beta-agonists.

- Inhaled magnesium sulphate is currently not recommended for the treatment of acute asthma.

- Intravenous magnesium sulphate (2 g IV slowly = 8 mmol) may be useful in patients with acute severe asthma (PEF < 50 % best or predicted) who have not had a good initial response to inhaled bronchodilator therapy. The most commonly reported adverse effects are flushing, fatigue, nausea, headache and hypotension.

- Early use of systemic corticosteroids for acute asthma significantly reduces hospital admission rates. Although there is no difference in clinical effects between oral and IV formulations of corticosteroids, the IV route is preferable because patients with near-fatal asthma may vomit or be unable to swallow.

- Consider intravenous salbutamol in patients unresponsive to nebulised therapy or where nebulised/inhaled therapy is not possible (e.g. a patient receiving bag-mask ventilation). Give as either a slow IV injection (250 mcg IV slowly) or continuous infusion of 3-20 mcg min^{-1}.

- There is no evidence of benefit and a higher incidence of adverse effects for intravenous aminophylline compared with standard care alone. If after obtaining senior advice the decision is taken to administer IV aminophylline a loading dose of 5 mg kg^{-1} is given over 20-30 min (unless on maintenance therapy), followed by an infusion of 500-700 mcg kg^{-1}h^{-1}. Serum theophylline concentrations should be maintained below 20 mcg ml^{-1} to avoid toxicity.

- These patients are often dehydrated or hypovolemic and will benefit from fluid replacement. Beta-2 agonists and steroids may induce hypokalaemia, which should be corrected with electrolyte supplements.

- Patients that fail to respond to initial treatment, or develop signs of life-threatening asthma, must be assessed by an intensive care specialist. These patients may benefit from tracheal intubation and ventilatory support. Routine use of non-invasive ventilation is not recommended.

- Sometimes it may be difficult to distinguish severe life-threatening asthma from anaphylaxis. Treat patients presenting with severe 'asthma-like' symptoms, but without pre-existing pulmonary disease (asthma, COPD), as if the cause was anaphylaxis. In these circumstances, administration of adrenaline 0.5 mg IM according to the anaphylaxis guidelines may be appropriate.

Cardiac arrest

- Follow standard BLS and ALS protocols. Ventilation will be difficult because of increased airway resistance; try to avoid gastric inflation.

- Intubate the trachea early. There is a significant risk of gastric inflation and hypoventilation of the lungs when attempting to ventilate a severe asthmatic without a tracheal tube.

- Respiratory rates of 8-10 breaths per minute and a tidal volume required for a normal chest rise during CPR should minimise dynamic hyperinflation of the lungs (air trapping).

- If dynamic hyperinflation of the lungs is suspected during CPR, compression of the chest wall and/or a period of apnoea (disconnection of tracheal tube) may relieve gas-trapping. Although this procedure is supported by limited evidence, it is unlikely to be harmful in an otherwise desperate situation.

- Dynamic hyperinflation increases transthoracic impedance, but modern impedance-compensated biphasic defibrillation waveforms are no less effective in patients with a higher impedance. As with standard ALS defibrillation protocols, consider increasing defibrillation energy if the first shock is unsuccessful.

- Look for reversible causes using the 4 Hs and 4 Ts approach.

- Tension pneumothorax can be difficult to diagnose in cardiac arrest; it may be indicated by unilateral expansion of the chest wall, shifting of the trachea, and subcutaneous emphysema. Pleural ultrasound in skilled hands is faster and more sensitive than chest X-ray for the detection of pneumothorax. Early needle decompression (thoracocentesis) followed by chest drain insertion is needed. Needle decompression may fail due to inadequate needle length. In the ventilated patient, thoracostomy (a surgical hole in the chest wall and pleura) may be quicker to do and more effective for decompressing the pneumothorax *(see trauma section)*.

- Always consider bilateral pneumothoraces in asthma-related cardiac arrest.

- Follow standard guidelines for post-resuscitation care.

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8. Anaphylaxis

8.1. Definition

Anaphylaxis is a severe, life-threatening, generalised or systemic hypersensitivity reaction.

This is characterised by rapidly developing life-threatening airway and/or breathing and/or circulation problems usually associated with skin and mucosal changes.

There are a number of national guidelines available all over Europe. The European Academy of Allergy and Clinical Immunology's (EAACI) Taskforce on Anaphylaxis defined clinical criteria for diagnosing anaphylaxis in 2014 (For more details see *http://www.eaaci.org/ resources/scientific-output/guidelines*).

8.2.   Aetiology

Anaphylaxis usually involves the release of inflammatory mediators from mast cells or basophils triggered by an allergen interacting with cell-bound immunoglobulin E (IgE). Non-IgE-mediated or non-immune release of mediators can also occur. Histamine and other inflammatory mediator release are responsible for the vasodilatation, oedema and increased capillary permeability.

Anaphylaxis can be triggered by any of a very broad range of triggers with food, drugs, stinging insects, and latex the most commonly identified triggers. Food is the commonest trigger in children and drugs the commonest in adults. Virtually any food or drug can be implicated, but certain foods (nuts) and drugs (muscle relaxants, antibiotics, nonsteroidal anti-inflammatory drugs and aspirin) cause most reactions. A significant number of cases of anaphylaxis are idiopathic.

The risk of death is increased in those with pre-existing asthma, particularly if the asthma is poorly controlled or in those asthmatics who fail to use, or delay treatment with, adrenaline.

When anaphylaxis is fatal, death usually occurs very soon after contact with the trigger. Fatal food reactions cause respiratory arrest typically after 30-35 min; insect stings cause collapse from shock after 10-15 min; and deaths caused by intravenous medication occurred most commonly within 5 min. Death rarely occurred more than six hours after contact with the trigger.

8.3.   Recognition

- Anaphylaxis is likely if a patient who is exposed to a trigger (allergen) develops a sudden illness (usually within minutes of exposure) with rapidly progressing skin changes and life-threatening airway and/or breathing and/or circulation problems. The reaction is usually unexpected.

- The lack of any consistent clinical manifestation and a range of possible presentations cause diagnostic difficulty. Patients have been given injections of adrenaline inappropriately for allergic reactions just involving the skin, or for vasovagal reactions or panic attacks. Guidelines for the treatment of an anaphylactic reaction must therefore take into account some inevitable diagnostic errors, with an emphasis on the need for safety.

Anaphylaxis is likely when all of the following three criteria are met:

- sudden onset and rapid progression of symptoms

- life-threatening **A**irway and/or **B**reathing and/or **C**irculation problems

- skin and/or mucosal changes (flushing, urticaria, angioedema)

The following supports the diagnosis:

- exposure to a known allergen for the patient

Remember:

- Skin or mucosal changes alone are not a sign of anaphylaxis.

- Skin and mucosal changes can be subtle or absent in up to 20 % of reactions (some patients can have only a decrease in blood pressure, i.e. a Circulation problem).

- There can also be gastrointestinal symptoms (e.g. vomiting, abdominal pain, incontinence).

8.3.1. Sudden onset and rapid progression of symptoms:

- The patient will feel and look unwell.

- Most reactions occur over several minutes. Rarely, reactions may be slower in onset.

- An intravenous trigger will cause a more rapid onset of reaction than stings, which, in turn, tend to cause a more rapid onset than orally ingested triggers.

- The patient is usually anxious and can experience a "sense of impending doom".

8.3.2. Life-threatening <u>A</u>irway, <u>B</u>reathing and <u>C</u>irculation problems:

Use the ABCDE approach to recognise these.

Airway problems:

- Airway swelling, e.g. throat and tongue swelling (pharyngeal/laryngeal oedema). The patient has difficulty in breathing and swallowing and feels that the throat is closing up.

- Hoarse voice.

- Stridor - this is a high-pitched inspiratory noise caused by upper airway obstruction.

Breathing problems:

- shortness of breath - increased respiratory rate

- wheeze

- patient becoming tired

- confusion caused by hypoxia

- cyanosis - this is usually a late sign

- respiratory arrest.

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Circulation problems:

- signs of shock - pale, clammy

- tachycardia

- hypotension - feeling faint, collapse

- decreased conscious level or loss of consciousness

- anaphylaxis can cause myocardial ischaemia and electrocardiograph (ECG) changes even in individuals with normal coronary arteries

- cardiac arrest

Circulation problems (often referred to as anaphylactic shock) can be caused by direct myocardial depression, vasodilation and capillary leak, and loss of fluid from the circulation.

The above Airway, Breathing and Circulation problems can all alter the patient's neurological status (**D**isability problems) because of decreased brain perfusion. There may be confusion, agitation and loss of consciousness.

8.3.3. Skin and, or mucosal changes:

These should be assessed as part of the Exposure when using the ABCDE approach.

- They are often the first feature and present in over 80 % of anaphylactic reactions.

- They can be subtle or dramatic.

- There may be just skin, just mucosal, or both skin and mucosal changes.

- There may be erythema - a patchy, or generalised, red rash.

- There may be urticaria (also called hives, nettle rash, weals or welts), which can appear anywhere on the body. The weals may be pale, pink or red, and may look like nettle stings. They can be different shapes and sizes, and are often surrounded by a red flare. They are usually itchy.

- Angioedema is similar to urticaria but involves swelling of deeper tissues, most commonly in the eyelids and lips, and sometimes in the mouth and throat.

Although skin changes can be worrying or distressing for patients and those treating them, skin changes without life-threatening airway, breathing or circulation problems do not signify anaphylaxis.

8.4. Differential diagnosis

8.4.1. Life-threatening conditions:

- Sometimes an anaphylactic reaction can present with symptoms and signs that are very similar to life-threatening asthma.

- A low blood pressure (or normal in children) with a petechial or purpuric rash can be a sign of septic shock. Seek help early if there are any doubts about the diagnosis and treatment.

- Following the ABCDE approach will help with treating the differential diagnoses.

8.4.2. Non life-threatening conditions (these usually respond to simple measures):

- faint (vasovagal episode)

- panic attack

- breath-holding episode in child

- idiopathic (non-allergic) urticaria or angioedema

There can be confusion between an anaphylactic reaction and a panic attack. Victims of previous anaphylaxis may be particularly prone to panic attacks if they think they have been re-exposed to the allergen that caused a previous problem. The sense of impending doom and breathlessness leading to hyperventilation are symptoms that resemble anaphylaxis in some ways. While there is no hypotension, pallor, wheeze, or urticarial rash or swelling, there may sometimes be flushing or blotchy skin associated with anxiety adding to the diagnostic difficulty. Diagnostic difficulty may also occur with vasovagal attacks after immunisation procedures, but the absence of rash, breathing difficulties, and swelling are useful distinguishing features, as is the slow pulse of a vasovagal attack compared with the rapid pulse of a severe anaphylactic episode. Fainting will usually respond to lying the patient down and raising the legs.

8.5. Treatment

As the diagnosis of anaphylaxis is not always obvious, all those who treat anaphylaxis must use the systematic ABCDE approach to the sick patient. Treat life-threatening problems as you find them. The key steps are described in the anaphylaxis algorithm *(figure 12.5)*.

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- All patients should be placed in a comfortable position. Patients with airway and breathing problems may prefer to sit up as this will make breathing easier. Lying flat with or without leg elevation is helpful for patients with a low blood pressure (Circulation problem). If the patient feels faint, do not sit or stand them up - this can cause cardiac arrest. Patients who are breathing and unconscious should be placed on their side (recovery position).

- Remove the trigger for an anaphylactic reaction if possible. Remove the stinger after a bee/wasp sting. Stop any drug suspected of causing an anaphylactic reaction (e.g. stop intravenous colloids or antibiotic). Do not delay definitive treatment if removing the trigger is not feasible.

- Monitor all patients who have suspected anaphylaxis as soon as possible (e.g. ambulance crew, emergency department). Minimum monitoring includes pulse oximetry, non-invasive blood pressure and a 3-lead ECG.

- Give the highest concentration of oxygen possible during resuscitation (usually greater than 10 l min^{-1}).

- Adrenaline is the most important drug for the treatment of an anaphylactic reaction. As an alpha-receptor agonist, it reverses peripheral vasodilation and reduces oedema. Its beta-receptor activity dilates the bronchial airways, increases the force of myocardial contraction, and suppresses histamine and leukotriene release. Adrenaline works best when given early after the onset of the reaction but it is not without risk, particularly when given intravenously. Adverse effects are extremely rare with correct doses injected intramuscularly (IM). The subcutaneous or inhaled routes for adrenaline are not recommended for the treatment of anaphylaxis because they are less effective than the IM route.

- The intramuscular (IM) route is the best for most individuals who have to give adrenaline to treat an anaphylactic reaction. Monitor the patient as soon as possible (pulse, blood pressure, ECG, pulse oximetry). This will help monitor the response to adrenaline.

- For adults and children > 12 years give an initial IM adrenaline dose of 0.5 mg (0.5 ml of 1:1000 adrenaline = 0.5 mg = 500 mcg). Further doses can be given at about 5-min intervals according to the patient's response.

- The best site for IM injection is the anterolateral aspect of the middle third of the thigh. The needle used for injection needs to be sufficiently long to ensure that the adrenaline is injected into muscle.

- The use of IV adrenaline applies only to specialists in the use and titration of vasopressors in their normal clinical practice (e.g. anaesthetists, emergency physicians, intensive care doctors). In patients with a spontaneous circulation, intravenous adrenaline can cause life-threatening hypertension, tachycardia, arrhythmias, and myocardial ischaemia. Patients who are given IV adrenaline must be monitored - continuous ECG and pulse oximetry and frequent non-invasive blood pressure measurements as a minimum.

- Titrate IV adrenaline using 50 mcg boluses according to response. If repeated adrenaline doses are needed, start an IV adrenaline infusion. The pre-filled 10 ml syringe of 1:10,000 adrenaline contains 100 mcg ml^{-1}. A dose of 50 mcg is 0.5 ml, which is the smallest dose that can be given accurately. Do not give the undiluted 1:1000 adrenaline concentration IV.

- Auto-injectors are often given to patients at risk of anaphylaxis for their own use. Healthcare professionals should be familiar with the use of the most commonly available auto-injector devices. If an adrenaline auto-injector is the only available adrenaline preparation when treating anaphylaxis, healthcare providers should use it.

- Give a rapid IV fluid challenge (500-1000 ml in an adult) and monitor the response; give further doses as necessary. There is no evidence to support the use of colloids over crystalloids in this setting. Consider colloid infusion as a cause in a patient receiving a colloid at the time of onset of an anaphylactic reaction and stop the infusion. Hartmann's solution or 0.9 % saline are suitable fluids for initial resuscitation. A large volume of fluid may be needed.

- Antihistamines are a second line treatment for an anaphylactic reaction. Give chlorphenamine 10 mg IM or IV slowly. Antihistamines (H1-antihistamine) may help counter histamine-mediated vasodilation and bronchoconstriction. Used alone, they are unlikely to be life-saving in a true anaphylactic reaction. There is little evidence to support the routine use of an H_2-antihistamine (e.g. ranitidine, cimetidine) for the initial treatment of anaphylaxis.

- Corticosteroids may help prevent or shorten protracted reactions. There is little evidence on which to base the optimum dose of hydrocortisone in anaphylaxis.

- The presenting symptoms and signs of severe anaphylaxis and life-threatening asthma can be the same. Consider further bronchodilator therapy with salbutamol (inhaled or IV), ipratropium (inhaled), aminophylline (IV) or magnesium (IV) *(see asthma)*. IV magnesium is a vasodilator and can make hypotension worse.

- Adrenaline remains the first line vasopressor for the treatment of anaphylactic reactions. Consider other vasopressors and inotropes (e.g. noradrenaline, vasopressin, terlipressin) when initial resuscitation with adrenaline and fluids has not been successful. Only use these drugs in specialist settings (e.g. intensive care units) where there is experience in their use. Glucagon can be useful to treat an anaphylactic reaction in a patient taking a beta-blocker.

- Cardiac arrest with suspected anaphylaxis should be treated with standard doses of IV or intraosseous (IO) adrenaline for cardiac arrest. If this is not feasible, consider IM adrenaline if cardiac arrest is imminent or has just occurred. Consider large volumes of intravenous fluids.

- Airway obstruction may occur rapidly in severe anaphylaxis, particularly in patients with angioedema. Warning signs are swelling of the tongue and lips, hoarseness and oropharyngeal swelling.

- Consider early tracheal intubation; delay may make intubation extremely difficult. As airway obstruction progresses, supraglottic airway devices (e.g. LMA) are likely to be difficult to insert. Attempts at tracheal intubation may exacerbate laryngeal oedema. Early involvement of a senior anaesthetist is mandatory when managing these patients. A surgical airway may be required if tracheal intubation is not possible.

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Figure 12.5
Anaphylaxis algorithm

Anaphylactic reaction?

Assess using ABCDE approach

Diagnosis - look for:
- Acute onset of illness
- Life-threatening Airway and/or Breathing and/or Circulation problems[1]
- And usually skin changes

- **Call for help**
- Lie patient flat with raised legs (if breathing allows)

Adrenaline[2]

When skills and equipment available:
- Establish airway
- High flow oxygen
- IV fluid challenge[3]
- Chlorphenamine[4]
- Hydrocortisone[5]

Monitor:
- Pulse oximetry
- ECG
- Blood pressure

[1] Life-threatening problems:
Airway: swelling, hoarseness, stridor
Breathing: rapid breathing, wheeze, fatigue, cyanosis, SpO$_2$ < 92 %, confusion
Circulation: pale, clammy, low blood pressure, faintness, drowsy/coma

[2] Adrenaline (give IM unless experienced with IV adrenaline)
IM doses of 1:1000 adrenaline (repeat after 5 min if no better)
- Adult   500 mcg IM (0.5 ml)
- Child more than 12 years   500 mcg IM (0.5 ml)
- Child 6-12 years   300 mcg IM (0.3 ml)
- Child less than 6 years   150 mcg IM (0.15 ml)

Adrenaline IV to be given **only by experienced specialists**
Titrate: Adults 50 mcg; Children 1 mcg kg^{-1}

[3] IV fluid challenge (crystalloid):
Adult   500-1000 ml
Child   20 ml kg^{-1}

Stop IV colloid if this might be the cause of anaphylaxis

	[4] Chlorphenamine (IM or slow IV)	[5] Hydrocortisone (IM or slow IV)
Adult or child more than 12 years	10 mg	200 mg
Child 6 - 12 years	5 mg	100 mg
Child 6 months to 6 years	2.5 mg	50 mg
Child less than 6 months	250 mcg kg^{-1}	25 mg

8.6. Investigations

The specific test to help confirm a diagnosis of an anaphylactic reaction is measurement of mast cell tryptase. In anaphylaxis, mast cell degranulation leads to markedly increased blood tryptase concentrations.

8.6.1. Mast cell tryptase sample timing

The time of onset of the anaphylactic reaction is the time when symptoms were first noticed.

a) Minimum: one sample at 1-2 h after the start of symptoms.

b) Ideally: Three timed samples:

1) Initial sample as soon as feasible after resuscitation has started - do not delay resuscitation to take sample.

2) Second sample at 1-2 h after the start of symptoms.

3) Third sample either at 24 h or in convalescence. This provides baseline tryptase levels - some individuals have an elevated baseline level.

c) Use a serum or clotted blood ('liver function test' bottle) sample.

d) Record the timing of each sample accurately on the sample bottle and request form.

e) Consult your local laboratory if you have any queries.

8.6.2. Discharge and follow-up

Patients who have had a suspected anaphylactic reaction should be treated and then observed for at least 6 h in a clinical area with facilities for treating life-threatening ABC problems. Patients with a good response to initial treatment should be warned of the possibility of an early recurrence of symptoms and in some circumstances should be kept under observation for up to 24 h (e.g. individuals with severe asthma or with a severe asthmatic component, patients presenting in the evening or at night, patients in areas where access to emergency care is difficult, previous history of biphasic reactions, possibility of continuing absorption of allergen).

The exact incidence of biphasic reactions is unknown. There is no reliable way of predicting who will have a biphasic reaction. It is therefore important that decisions about discharge are made for each patient by an experienced clinician.

Before discharge from hospital all patients with anaphylaxia must be:

• Given clear instructions to return to hospital if symptoms return.

• Considered for an adrenaline auto-injector, or given a replacement and ensured that appropriate training has been given.

• Have a plan for follow-up, including contact with the patient's general practitioner.

• Referred to an allergy specialist to identify the cause, and thereby reduce the risk of future reactions and prepare the patient to manage future episodes themselves.

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9. Cardiac arrest following cardiac surgery

After major cardiac surgery, cardiac arrest is relatively common in the immediate post-operative phase, with a reported incidence of 0.7 %-8 %. Cardiac arrest is usually preceded by physiological deterioration, although it may occur suddenly in stable patients. Continuous monitoring on the intensive care unit (ICU) enables immediate intervention at the time of arrest.

Key to the successful resuscitation of cardiac arrest in these patients is recognition of the need to perform emergency resternotomy early, especially in the context of tamponade or haemorrhage, where external chest compressions may be ineffective.

9.1. Aetiology

There are usually specific causes of cardiac arrest that are all potentially reversible. The main causes of cardiac arrest in the initial post-operative period include:

- cardiac tamponade

- myocardial ischaemia

- hypovolaemia

- pacing failure

- tension pneumothorax

9.2. Diagnosis

An immediate decision on the likely cause of cardiac arrest must be made to enable rapid intervention and successful resuscitation. Patients in the ICU are highly monitored and an arrest is most likely to be signalled by monitoring alarms where absence of pulsation or perfusing pressure on the arterial line, loss of pulse oximeter trace, pulmonary artery (PA) trace, or end-tidal CO_2 trace and rapid assessment of the patient can be sufficient to indicate cardiac arrest without the need to palpate a central pulse. Call for senior help early including a cardiothoracic surgeon and cardiac anaesthetist.

9.3. Treatment

- Chest compressions can cause sternal disruption or cardiac damage in the post-cardiac surgery setting. If VF is diagnosed, immediately administer external defibrillation. A witnessed and monitored VF/pVT cardiac arrest should be treated immediately with up to three quick successive (stacked) defibrillation attempts. Three failed shocks should trigger the need for emergency resternotomy. Further defibrillation is attempted as indicated in the universal algorithm and should be performed with internal paddles at 20 J if resternotomy has been performed.

- In asystole immediately establish emergency temporary pacing at maximum amplitude.

- In PEA, turn the pacing off to verify there is no underlying VF, which must be treated by defibrillation.

- Otherwise start external chest compressions immediately in patients who arrest with monitoring indicating no output. Verify the effectiveness of compressions by looking at the arterial trace, aiming to achieve a systolic blood pressure > 60 mmHg and a diastolic blood pressure > 25 mmHg at a rate of 100-120 min^{-1}. Inability to obtain this goal with external chest compressions indicates that cardiac tamponade or extreme hypovolaemia is likely and emergency resternotomy should be performed.

- Consider reversible causes:

    - **Hypoxia** – check tracheal tube position, ventilate with 100 % oxygen.

    - **Tension pneumothorax** – check tracheal position, listen for air entry.

    - **Pacing failure** – check pacing box output and pacing wire integrity. In asystole, secondary to a loss of cardiac pacing, chest compressions may be delayed momentarily as long as the surgically inserted temporary pacing wires can be connected rapidly and pacing re-established (DDD at 100 min^{-1} at maximum amplitude).

- Use adrenaline very cautiously and titrate to effect (IV doses of up to 100 mcg in adults). Consider amiodarone 300 mg in patients with refractory shockable rhythms (VF/pVT), but do not delay resternotomy.

- Emergency resternotomy is an integral part of resuscitation after cardiac surgery, once all other reversible causes have been excluded. Once an adequate airway and ventilation has been established, and if three attempts at defibrillation have failed in VF/pVT, undertake resternotomy without delay. Emergency resternotomy is also indicated in asystole or PEA, when other treatments have failed. Resuscitation teams should be well rehearsed in this technique so that it can be performed safely within 5 min of the onset of cardiac arrest by anyone with appropriate training.

- The same strategy is appropriate for patients following non-sternotomy cardiac surgery, but surgeons performing these operations should have already made clear their instructions for chest reopening in an arrest.

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10. Traumatic cardiac arrest

Traumatic cardiac arrest (TCA) carries a very high mortality, but in those where ROSC can be achieved, neurological outcome in survivors appears to be much better than in other causes of cardiac arrest. A large systematic review reported an overall survival rate of 3.3 % in blunt and 3.7 % in penetrating trauma, with good neurological outcome in 1.6 % of all cases.

It is vital that a medical cardiac arrest is not misdiagnosed as a TCA and must be treated with the universal ALS algorithm. Cardiac arrest or other causes of sudden loss of consciousness (e.g. hypoglycaemia, stroke, seizures) may cause a secondary traumatic event. Some observational studies have reported that about 2.5 % of non-traumatic OHCAs occur in cars. In these cases, shockable rhythms (VF/pVT) are more common. The primary cause of the cardiac arrest can be elucidated from information about past medical history, events preceding the accident (if possible), and a systematic post-ROSC assessment, including a 12-lead ECG.

Commotio cordis is actual or near cardiac arrest caused by a blunt impact to the chest wall over the heart. A blow to the chest can cause VF if the striking object strikes the chest within a 20 ms window of the upstroke of the T-wave. Commotio cordis occurs mostly during sports and recreational activities and victims are usually teenage males. Follow standard CPR guidelines. Early defibrillation is important for survival.

10.1. Diagnosis

The diagnosis of traumatic cardiac arrest is made clinically. The patient presents with agonal or absent spontaneous respiration and absence of a central pulse.

A peri-arrest state is characterised by cardiovascular instability, hypotension, loss of peripheral pulses in uninjured regions and a deteriorating conscious level without obvious central nervous system (CNS) cause. If untreated, this state is likely to progress to cardiac arrest.

- The response to TCA is time-critical and success depends on a well-established chain of survival, including advanced prehospital and specialised trauma centre care. Immediate resuscitative efforts in TCA focus on simultaneous treatment of reversible causes, which takes priority over chest compressions. Figure 12.6 shows a traumatic cardiac (peri-) arrest algorithm.

- Chest compressions are still the standard of care in patients with cardiac arrest, irrespective of aetiology. In cardiac arrest caused by hypovolaemia, cardiac tamponade or tension pneumothorax, chest compressions are unlikely to be as effective as in normovolaemic cardiac arrest. Because of this fact, chest compressions take a lower priority than the immediate treatment of reversible causes, e.g. thoracotomy, controlling haemorrhage etc.

- In an out-of-hospital setting, only essential life-saving interventions should be performed on scene followed by rapid transfer to the nearest appropriate hospital.

- If available, ultrasound will help diagnose rapidly haemoperitoneum, haemopneumothorax, tension pneumothorax and cardiac tamponade. This requires a trained operator and should not delay treatment.

- Treatment of reversible causes:
    - **Hypoxaemia** - Effective airway management and ventilation can reverse hypoxic cardiac arrest and it is essential to establish and maintain oxygenation of trauma patients with a severely compromised airway. Tracheal intubation in trauma patients is a difficult procedure with a high failure rate if carried out by less experienced care providers. Use basic airway manoeuvres and second-generation supraglottic airways to maintain oxygenation if tracheal intubation cannot be accomplished immediately.

10.2. Treatment

Figure 12.6
Traumatic cardiac (peri-) arrest algorithm
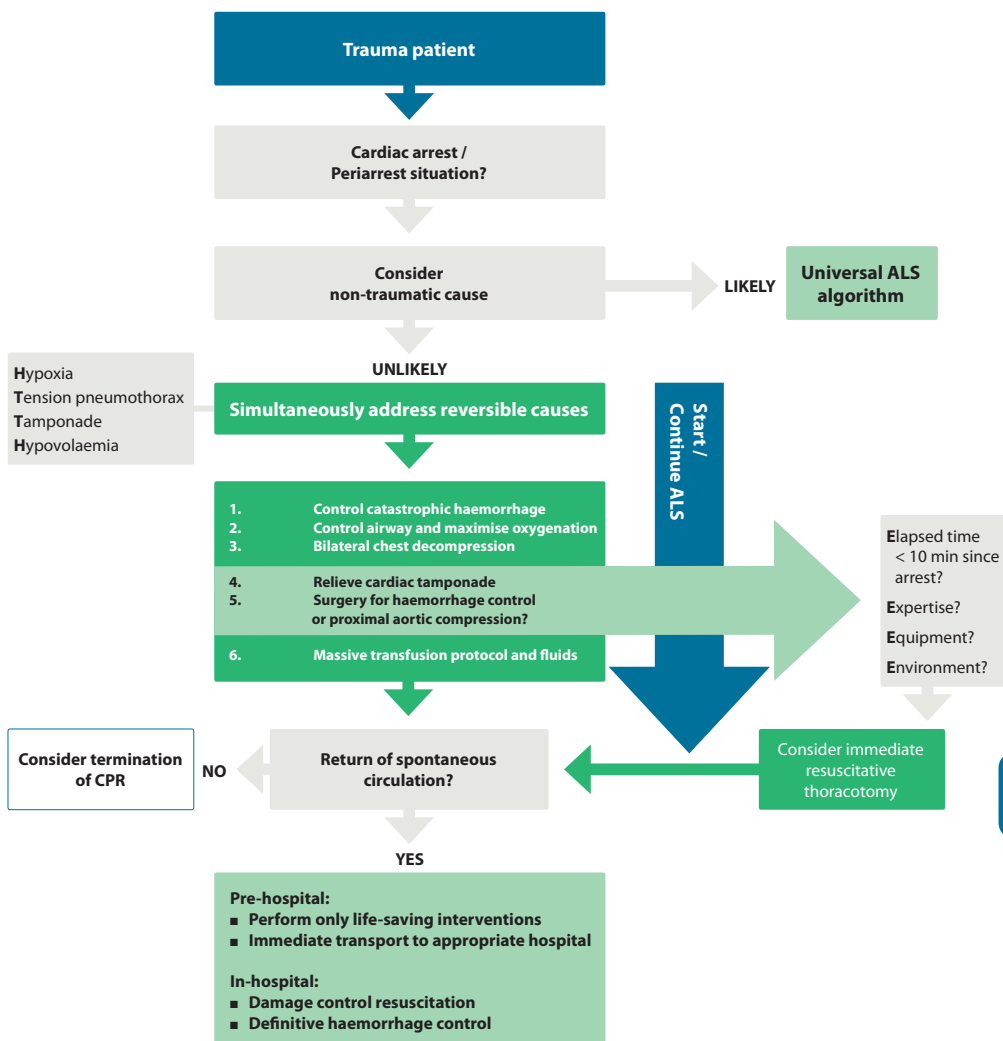
- **Hypovolaemia** - Uncontrolled haemorrhage is the cause of traumatic cardiac arrest in 48 % of all TCA. The treatment of severe hypovolaemic shock has several elements. The main principle is to achieve 'haemostasis without delay', usually with surgical or radiological intervention. Temporary haemorrhage control can be lifesaving. Treat compressible external haemorrhage with direct pressure (with or without a dressing), use tourniquets if needed and/or apply

topical haemostatic agents. Non-compressible haemorrhage is more difficult. Use splints (pelvic splint), blood products, intravenous fluids and tranexamic acid while moving the patient to surgical haemorrhage control.

- **Tension pneumothorax** - Perform bilateral thoracostomies in the 4th intercostal space to decompress the chest during TCA. In the presence of positive pressure ventilation, thoracostomies are likely to be more effective than needle thoracocentesis and quicker than inserting a chest tube.

- **Cardiac tamponade** - Where there is TCA and penetrating trauma to the chest or epigastrium, immediate resuscitative thoracotomy (RT) (via a clamshell incision can be life saving. Needle aspiration of tamponade, with or without ultrasound guidance, is unreliable because the pericardium is commonly full of clotted blood. If thoracotomy is not possible, consider ultrasound guided pericardiocentesis to treat TCA associated with suspected cardiac tamponade. Non-image guided pericardiocentesis is an alternative only if ultrasound is not available.

- Positive pressure ventilation worsens hypotension by impeding venous return to the heart, particularly in hypovolaemic patients. Low tidal volumes and slow respiratory rates may help optimise cardiac preload. Monitor ventilation with continuous waveform capnography and adjust to achieve normocapnia.

- Over the past ten years the principle of 'damage control resuscitation' has been adopted in trauma resuscitation for uncontrolled haemorrhage. Damage control resuscitation combines permissive hypotension and haemostatic resuscitation with damage control surgery. Limited evidence and general consensus have supported a conservative approach to intravenous fluid infusion, with permissive hypotension until surgical haemostasis is achieved. Permissive hypotension allows intravenous fluid administration to a volume sufficient to maintain a radial pulse. Haemostatic resuscitation is the very early use of blood products as primary resuscitation fluid to prevent exsanguination by trauma-induced coagulopathy. The recommended ratio of packed red cells, fresh frozen plasma and platelets is 1:1:1.

- Tranexamic acid (TXA) (loading dose 1 g over 10 min followed by infusion of 1 g over 8 h) increases survival from traumatic haemorrhage. It is most effective when administered within the first hour and certainly within the first three hours following trauma.

Resuscitative thoracotomy

- Succesful RT is time critical. One UK service recommends that if surgical intervention cannot be accomplished within 10 min after loss of pulse in patients with penetrating chest injury, on scene RT should be considered.

The prerequisites for a successful RT can be summarised as the 'four Es rule' (4E):

- **Expertise:** teams must be led by a highly trained and competent healthcare practitioner.

- **Equipment:** adequate equipment to carry out RT and to deal with the intrathoracic findings is mandatory.

- **Environment:** ideally RT should be carried out in an operating theatre. RT should not be carried out if there is inadequate physical access to the patient, or if the receiving hospital is not easy to reach.

- **Elapsed time:** the time from loss of vital signs to commencing a RT should not be longer than 10 minutes.

If any of the four criteria is not met, RT is futile and exposes the team to unnecessary risks.

Consider emergency department thoracotomy in the following circumstances:

- blunt trauma patients with less than 10 min of prehospital CPR;
- penetrating torso trauma patients with less than 15 min of CPR.

11. Pregnancy

Mortality related to pregnancy is relatively rare in Europe (estimate 16 per 100,000 live births) although there is a large variation between countries. The fetus must always be considered when an adverse cardiovascular event occurs in a pregnant woman. Fetal survival usually depends on maternal survival and initial resuscitation efforts should focus on the pregnant mother.

Significant physiological changes occur during pregnancy; for example, cardiac output, circulatory volume, minute ventilation, and oxygen consumption all increase. The gravid uterus can cause compression of iliac and abdominal vessels when the mother is in the supine position, resulting in reduced cardiac output and hypotension.

11.1. Causes of cardiac arrest in pregnancy

Cardiac arrest in pregnancy is most commonly caused by:

- cardiac disease
- pulmonary embolism
- psychiatric disorders
- hypertensive disorders of pregnancy
- sepsis
- haemorrhage
- amniotic fluid embolism
- ectopic pregnancy.

Pregnant women can also have the same causes of cardiac arrest as females of the same age group (e.g. anaphylaxis, drug overdose, trauma).

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11.2. Treatment

11.2.1. Prevention of cardiac arrest in pregnancy

In an emergency, use the ABCDE approach. Many cardiovascular problems associated with pregnancy are caused by compression of the inferior vena cava. Treat a distressed or compromised pregnant patient as follows:

- Place the patient in the left lateral position or manually and gently displace the uterus to the left.

- Give high-flow oxygen guided by pulse oximetry.

- Give a fluid bolus if there is hypotension or evidence of hypovolaemia.

- Immediately re-evaluate the need for any drugs being given.

- Seek expert help early. Obstetric and neonatal specialists should be involved early in the resuscitation.

- Identify and treat the underlying cause.

11.2.2. Modifications for cardiac arrest

- In cardiac arrest, all the principles of basic and advanced life support apply.

- Summon help immediately. For effective resuscitation of mother and fetus, expert help must be obtained; this should include an obstetrician and neonatologist.

- Start CPR according to standard guidelines. Ensure good quality chest compressions with minimal interruptions.

- After 20 weeks gestation the pregnant woman's uterus can press down against the inferior vena cava and the aorta, impeding venous return, cardiac output and uterine perfusion. Caval compression limits the effectiveness of chest compressions.

- Manually displace the uterus to the left to remove caval compression. The hand position for chest compressions may need to be slightly higher on the sternum for patients with advanced pregnancy e.g. third trimester.

- Add left lateral tilt if this is feasible and ensure the chest remains supported on a firm surface (e.g. in the operating room) – the optimal angle of tilt is unknown. Aim for between 15 and 30 degrees. Even a small amount of tilt may be better than no tilt. The angle of tilt used needs to enable high-quality chest compressions and if needed, allow Caesarean delivery of the fetus *(see below)*.

Methods for tilting include:

- if the patient is already on a spineboard or operating table the board or table can be tilted to provide a left lateral tilt;

    - sand bags, firm pillows, or a purpose made wedge;

    - using the thighs of kneeling rescuers to tilt the torso.

- Start preparing for emergency Caesarean section *(see below)* - the fetus will need to be delivered if initial resuscitation efforts fail.

- There is an increased risk of pulmonary aspiration of gastric contents in pregnancy. Early tracheal intubation decreases this risk, but can be more difficult in the pregnant patient. A tracheal tube 0.5-1 mm internal diameter (ID) smaller than that used for a non-pregnant woman of similar size may be necessary because of maternal airway narrowing from oedema and swelling. Expert help, a failed intubation drill, and the use of alternative airway devices may be needed.

- Attempt defibrillation using standard energy doses.

11.2.3. Reversible causes

Look for reversible causes using the 4 Hs and 4 Ts approach. Abdominal ultrasound by a skilled operator to detect possible causes during cardiac arrest can be useful. It can also permit an evaluation of fetal viability, multiple pregnancy and placental localisation. It should not however delay treatments. Specific reversible causes of cardiac arrest in pregnancy are:

- **Haemorrhage:** This can occur both antenatally and postnatally. Causes include ectopic pregnancy, placental abruption, placenta praevia and uterine rupture. Maternity units should have a massive haemorrhage protocol. Treatment is based on the ABCDE approach. The key step is to stop the bleeding. Consider the following: fluid resuscitation including use of a rapid transfusion system and cell salvage, correction of coagulopathy, oxytocin, ergometrine and prostaglandins to correct uterine atony, uterine compression sutures, intrauterine balloon devices, radiological embolisation of a bleeding vessel, and surgical control including aortic cross clamping/compression and hysterectomy. Placenta percreta may require extensive intra-pelvic surgery.

- **Drugs:** Overdose can occur in women with eclampsia receiving magnesium sulphate, particularly if the patient becomes oliguric. Give calcium to treat magnesium toxicity (see life-threatening electrolyte abnormalities). Central neural blockade for analgesia or anaesthesia can cause problems due to sympathetic blockade (hypotension, bradycardia) or local anaesthetic toxicity *(see poisoning section)*.

- **Cardiovascular disease:** Myocardial infarction and aneurysm or dissection of the aorta or its branches, and peripartum cardiomyopathy cause most deaths from acquired cardiac disease. Patients with known cardiac disease need to be managed in a specialist unit. Pregnant women may develop an acute coronary syndrome, typically in association with risk factors such as obesity, older age, higher parity, smoking, diabetes, pre-existing hypertension and a family history of ischaemic heart disease. Pregnant patients can have atypical features such as epigastric pain and vomiting. Percutaneous coronary intervention (PCI) is the reperfusion strategy of choice for ST- elevation myocardial infarction in pregnancy. Thrombolysis should be considered if urgent PCI is unavailable. Increasing numbers of women with congenital heart disease are becoming pregnant.

- **Pre-eclampsia and eclampsia:** Eclampsia is defined as the development of convulsions and/or unexplained coma during pregnancy or postpartum in patients with signs and

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symptoms of pre-eclampsia. Magnesium sulphate treatment may prevent eclampsia developing in labour or immediately postpartum in women with pre-eclampsia.

- **Amniotic fluid embolism** usually presents around the time of delivery often in the labouring mother with sudden cardiovascular collapse, breathlessness, cyanosis, arrhythmias, hypotension and haemorrhage associated with disseminated intravascular coagulopathy. Treatment is supportive based on the ABCDE approach and correction of coagulopathy. There is no specific therapy.

- **Pulmonary embolus** causing cardiopulmonary collapse can present throughout pregnancy. CPR should be started with modifications as necessary. The use of fibrinolysis (thrombolysis) needs considerable thought, particularly if a peri-mortem Caesarean section is being considered. If the diagnosis is suspected and maternal cardiac output has not returned it should be given.

11.2.4. Peri-mortem Caesarean section

When initial resuscitation attempts fail, delivery of the fetus may improve the chances of successful resuscitation of both the mother and fetus. The best survival rate for infants over 24-25 weeks gestation occurs when delivery of the infant is achieved within 5 min after the mother's cardiac arrest. This requires that Caesarean section starts at about 4 min after cardiac arrest. At older gestational ages (30-38 weeks), infant survival is possible even when delivery was after 5 minutes from the onset of maternal cardiac arrest. Delivery relieves caval compression and may improve the likelihood of resuscitating the mother by permitting an increase in venous return during the CPR attempt. Delivery also enables access to the abdominal cavity so that aortic clamping or compression is possible. Once the fetus has been delivered resuscitation of the newborn child can also begin.

In the supine position, the gravid uterus begins to compromise blood flow in the inferior vena cava and abdominal aorta at approximately 20 weeks' gestation; however, fetal viability currently begins at approximately 24 weeks.

- Gestational age < 20 weeks. Urgent Caesarean delivery need not be considered, because a gravid uterus of this size is unlikely to compromise maternal cardiac output and fetal viability is not an issue.

- Gestational age approximately 20-23 weeks. Initiate emergency delivery of the fetus to permit successful resuscitation of the mother, not survival of the delivered infant, which is unlikely at this gestational age.

- Gestational age approximately > 24 weeks. Initiate emergency delivery to help save the life of both the mother and the infant.

11.2.5. Planning for resuscitation in pregnancy

Advanced life support in pregnancy requires co-ordination of maternal resuscitation, Caesarean delivery of the fetus, and newborn resuscitation within 5 min. To achieve this, units likely to deal with cardiac arrest in pregnancy should:

- have in place plans and equipment for resuscitation of both the pregnant patient and newborn

- ensure early involvement of obstetric, anaesthetic and neonatal teams

- ensure regular training in obstetric emergencies

12. Obesity

Worldwide obesity has more than doubled since 1980. Many clinical studies have linked body mass index (BMI) to outcomes for a wide variety of cardiovascular and non-cardiovascular conditions. Traditional cardiovascular risk factors (hypertension, diabetes, lipid profile, prevalent coronary heart disease, heart failure, and left ventricular hypertrophy) are common in obese patient. Obesity is associated with increased risk of sudden cardiac death. Leading causes of death are dilated cardiomyopathy and severe coronary atherosclerosis.

12.1. Modifications to cardiopulmonary resuscitation

- Physical and physiological factors related to obesity may adversely affect the delivery of CPR, including patient access and transportation, patient assessment, difficult IV access, airway management, quality of chest compressions, the efficacy of vasoactive drugs, and the efficacy of defibrillation because none of these measures are standardized to a patient's weight.

- A patient's weight should be considered when organising prehospital resuscitation, especially with regards to technical support and number of ambulance crew members. More rescuers than usual may be required to assist in moving the patient and rescuer fatigue, particularly in relation to the delivery of chest compressions.

- Chest compressions are most effective when performed with the patient lying on a firm surface. It may be unsafe for the patient and rescuers to attempt to move the obese patient down onto the floor, but it is not always necessary because the heavier torso sinks into the mattress, leaving less potential for mattress displacement during chest compression.

- Rescuer fatigue may necessitate the need to change rescuers more frequently than the standard two minute interval. The slope of the anterior chest wall, thoracic dimensions and patient weight limits use of mechanical resuscitation devices.

- Defibrillation protocols for obese patients should follow standard recommendations. Consider higher shock energies for defibrillation if initial defibrillation attempts fail.

- Early tracheal intubation by an experienced provider removes the need for prolonged bag-mask ventilation, and may reduce any risk of aspiration. In all patients with extreme obesity, difficult intubation must be anticipated, with a clear failed intubation drill if necessary. If intubation fails, use of a supraglottic airway device (SAD) with oesophaegeal drainage tube is a suitable option.

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13. Electrocution

Electrical injury is a relatively infrequent but potentially devastating multi-system injury with high morbidity and mortality. Most electrical injuries in adults occur in the workplace and are associated generally with high voltage, whereas children are at risk primarily at home, where the voltage is lower (220 V in Europe, Australia, Asia; 110 V in the USA and Canada). Electrocution from lightning strikes is rare, but causes about 1000 deaths worldwide each year.

Factors influencing the severity of electrical injury include whether the current is alternating (AC) or direct (DC), voltage, magnitude of energy delivered, resistance to current flow, pathway of current through the patient, and the area and duration of contact. Skin resistance is decreased by moisture, which increases the likelihood of injury. Electric current follows the path of least resistance; conductive neurovascular bundles within limbs are particularly prone to damage.

Contact with AC may cause tetanic contraction of skeletal muscle, which may prevent release from the source of electricity. Myocardial or respiratory failure may cause immediate death:

- Respiratory arrest may be caused by central respiratory depression or paralysis of the respiratory muscles.

- Current may precipitate VF if it traverses the myocardium during the vulnerable period (analogous to an R-on-T phenomenon). Electrical current may also cause myocardial ischaemia because of coronary artery spasm.

- Asystole may be primary, or secondary to asphyxia following respiratory arrest.

Current that traverses the myocardium is more likely to be fatal. A transthoracic (hand to hand) pathway is more likely to be fatal than a vertical (hand to foot) or straddle (foot to foot) pathway. There may be extensive tissue destruction along the current pathway.

Lightning strikes deliver as much as 300 kV over a few milliseconds. Most of the current from a lightning strike passes over the surface of the body in a process called external flashover. Both industrial shocks and lightning strikes cause deep burns at the point of contact - in industry the points of contact are usually on the upper limbs, hands and wrists, whilst with lightning they are mostly on the head, neck and shoulders. Injury may also occur indirectly through ground current or current 'splashing' from a tree or other object that is hit by lightning. Explosive force generated by a lightning strike may cause blunt trauma.

Unique pattern of skin lesions called feathering or Lichtenberg figure is a pathognomonic symptom that is seen only in patients struck by lightning. Unconscious patients with linear or punctuate burns (feathering) should be treated as victims of lightning strike.

The pattern and severity of injury from a lightning strike varies considerably. As with industrial and domestic electric shock, death is caused by cardiac or respiratory arrest. In those who survive the initial shock, extensive catecholamine release or autonomic stimulation may occur, causing hypertension, tachycardia, nonspecific ECG changes

(including prolongation of the QT interval and transient T wave inversion), and myocardial necrosis. Creatine kinase may be released from myocardial and skeletal muscle. Lightning also causes various central and peripheral neurological problems.

13.1. Treatment

Ensure that any power source is switched off and do not approach the victim until it is safe. High voltage (above domestic mains) electricity can arc and conduct through the ground for up to a few metres around the victim. It is safe to approach and handle casualties after lightning strike, although it would be wise to move to a safer environment. Follow standard resuscitation guidelines.

- Airway management can be difficult if there are electrical burns around the face and neck. Intubate the trachea early in these cases as soft tissue oedema can cause subsequent airway obstruction. Immobilize the spine until evaluation can be performed.

- Muscular paralysis, especially after high voltage, may persist for several hours; ventilatory support is required during this period.

- Ventricular fibrillation is the commonest initial arrhythmia after high voltage AC shock; treat with prompt attempted defibrillation. Asystole is more common after DC shock; use standard guidelines for treatment of this and of other arrhythmias.

- Remove smouldering clothing and shoes to prevent further thermal injury.

- Vigorous fluid therapy is required if there is significant tissue destruction. Maintain a good urine output to increase excretion of myoglobin, potassium and other products of tissue damage.

- Consider early surgical intervention in patients with severe thermal injuries.

- Conduct a thorough secondary survey to exclude injuries caused by tetanic muscular contraction or from the person being thrown by the force of the shock.

- Electrocution can cause severe, deep soft tissue injury with relatively minor skin wounds because current tends to follow neurovascular bundles; look carefully for features of compartment syndrome, which will necessitate fasciotomy.

13.2. Further treatment and prognosis

Immediate resuscitation in young victims of cardiac arrest due to electrocution can result in survival. Successful resuscitation has been reported after prolonged life support. All those who survive electrical injury should be monitored in hospital if they have a history of cardiorespiratory problems or have suffered:

- loss of consciousness

- cardiac arrest

- electrocardiographic abnormalities

- soft tissue damage and burns

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Severe burns (thermal or electrical), myocardial necrosis, the extent of central nervous system injury, and secondary multiple system organ failure, determine the morbidity and long-term prognosis. Bone marrow embolism has also been reported in some cases. There is no specific therapy for electrical injury, and the management is symptomatic. Prevention remains the best way to minimise the prevalence and severity of electrical injury.

KEY LEARNING POINTS

- The conditions described in this chapter account for a large proportion of cardiac arrests in younger patients.

- Use the ABCDE approach for early recognition and treatment to prevent cardiac arrest.

- High-quality CPR and treatment of reversible causes is the mainstay of treatment of cardiac arrest from any cause.

- Call for expert help early when specialist procedures are needed - e.g. delivery of fetus for cardiac arrest in pregnancy.

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Chapter 13.

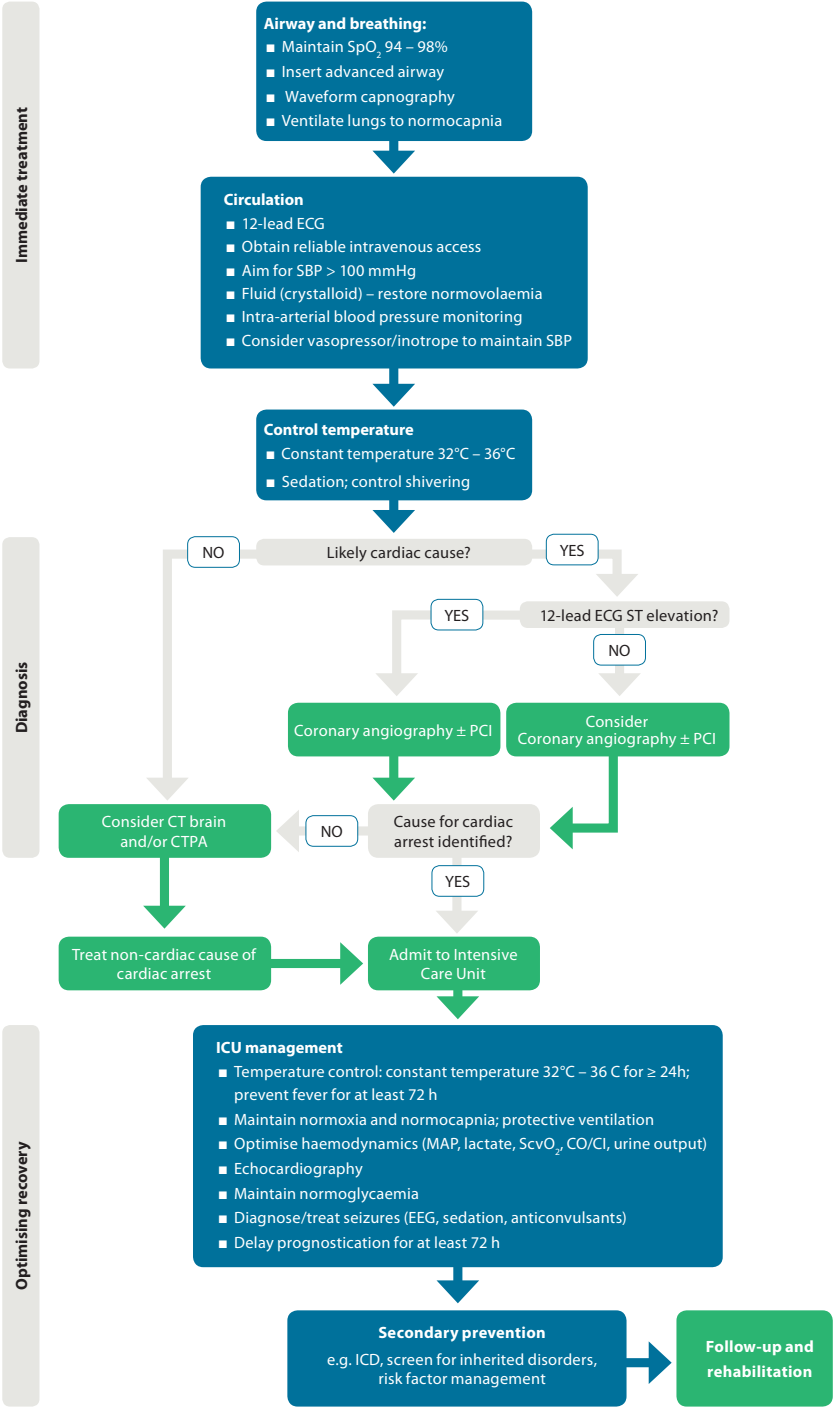# Post-resuscitation care

LEARNING OUTCOMES

To understand:

- the need for continued resuscitation after return of spontaneous circulation
- how to treat the post-cardiac arrest syndrome
- how to facilitate transfer of the patient safely
- the role and limitations of assessing prognosis after cardiac arrest

1.   Introduction

Successful return of spontaneous circulation (ROSC) is the first step toward the goal of complete recovery from cardiac arrest. The complex pathophysiological processes that occur following whole body ischaemia during cardiac arrest and the subsequent reperfusion response during CPR and following successful resuscitation have been termed the post-cardiac arrest syndrome. Depending on the cause of the arrest, and the severity of the post-cardiac arrest syndrome, many patients will require multiple organ support and the treatment they receive during this post-resuscitation period influences significantly the overall outcome and particularly the quality of neurological recovery. The post-resuscitation phase starts at the location where ROSC is achieved but, once stabilised, the patient is transferred to the most appropriatearea of high level care (e.g., emergency room, cardiac catheterisation laboratory or intensive care unit (ICU)) for continued diagnosis, monitoring and treatment. The post-resuscitation care algorithm *(figure 13.1)* outlines some of the key interventions required to optimise outcome for these patients. If there is any doubt about the patient´s neurological function, the patient's trachea should be intubated and treatment to optimise haemodynamic, respiratory and metabolic variables, together with targeted temperature management started, following the local standardised treatment plan.

Figure 13.1

Return of spontaneous circulation and comatose

Immediate treatment

Airway and breathing:
- Maintain SpO$_2$ 94 – 98%
- Insert advanced airway
- Waveform capnography
- Ventilate lungs to normocapnia

Circulation
- 12-lead ECG
- Obtain reliable intravenous access
- Aim for SBP > 100 mmHg
- Fluid (crystalloid) – restore normovolaemia
- Intra-arterial blood pressure monitoring
- Consider vasopressor/inotrope to maintain SBP

Control temperature
- Constant temperature 32°C – 36°C
- Sedation; control shivering

Diagnosis

Likely cardiac cause? — NO / YES

12-lead ECG ST elevation? — YES / NO

Coronary angiography ± PCI

Consider Coronary angiography ± PCI

Consider CT brain and/or CTPA — NO — Cause for cardiac arrest identified? — YES

Treat non-cardiac cause of cardiac arrest → Admit to Intensive Care Unit

Optimising recovery

ICU management
- Temperature control: constant temperature 32°C – 36 C for ≥ 24h; prevent fever for at least 72 h
- Maintain normoxia and normocapnia; protective ventilation
- Optimise haemodynamics (MAP, lactate, ScvO$_2$, CO/CI, urine output)
- Echocardiography
- Maintain normoglycaemia
- Diagnose/treat seizures (EEG, sedation, anticonvulsants)
- Delay prognostication for at least 72 h

Secondary prevention
e.g. ICD, screen for inherited disorders, risk factor management

→ **Follow-up and rehabilitation**

1.1.   The post-cardiac arrest syndrome

The post-cardiac arrest syndrome comprises post-cardiac arrest brain injury, post–cardiac arrest myocardial dysfunction, the systemic ischaemia/reperfusion response, and the persistent precipitating pathology. The severity of this syndrome will vary with the duration and cause of cardiac arrest. It may not occur at all if the cardiac arrest is brief. Post-cardiac arrest brain injury manifests as coma, seizures, myoclonus, varying degrees of neurocognitive dysfunction and brain death. Post-cardiac arrest brain injury may be exacerbated by microcirculatory failure, impaired autoregulation, hypercarbia, hypoxaemia and hyperoxaemia, pyrexia, hyperglycaemia and seizures. Significant myocardial dysfunction is common after cardiac arrest but typically recovers by 2-3 days. The whole body ischaemia/reperfusion that occurs with resuscitation from cardiac arrest activates immunological and coagulation pathways contributing to multiple organ failure and increasing the risk of infection. Thus, the post-cardiac arrest syndrome has many features in common with sepsis, including intravascular volume depletion and vasodilation.

2.   Continued resuscitation

In the immediate post-resuscitation phase, pending transfer to an appropriate area of high level care, treat the patient by following the ABCDE approach.

2.1.   Airway and breathing

2.1.1. Control of oxygenation

Patients who have had a brief period of cardiac arrest responding immediately to appropriate treatment may achieve an immediate return of normal cerebral function. These patients do not require tracheal intubation and ventilation but should be given oxygen via a facemask if their arterial oxygen saturation is less than 94 %. Hypoxaemia and hypercarbia both increase the likelihood of a further cardiac arrest and may contribute to secondary brain injury. Given the evidence of harm after myocardial infarction and the possibility of increased neurological injury after cardiac arrest, as soon as arterial blood oxygen saturation can be monitored reliably (by blood gas analysis and/or pulse oximetry), titrate the inspired oxygen concentration to maintain the arterial blood oxygen saturation in the range of 94-98 %. Avoid hyperoxaemia, which is also harmful – ensure reliable measurement of arterial oxygen saturation before reducing the inspired oxygen concentration.

2.1.2. Control of ventilation

Consider tracheal intubation, sedation and controlled ventilation in any patient with obtunded cerebral function. Ensure the tracheal tube is positioned correctly, well above the carina. Hypocarbia causes cerebral vasoconstriction and a decreased cerebral blood flow. After cardiac arrest, hypocapnia induced by hyperventilation causes cerebral ischaemia. Adjust ventilation to achieve normocapnia and monitor this using the end-tidal CO_2 and arterial blood gas values. Lowering the body temperature decreases the metabolism and may increase the risk of hypocapnia during the temperature intervention.

Although protective lung ventilation strategies have not been studied specifically in post-cardiac arrest patients, given that these patients develop a marked inflammatory response, it seems rational to apply protective lung ventilation: tidal volume 6-8 ml kg^{-1} ideal body weight and positive end expiratory pressure 4-8 cmH$_2$O. Insert a gastric tube to decompress the stomach; gastric distension caused by mouth-to-mouth or bag-mask-valve ventilation will splint the diaphragm and impair ventilation. Give adequate doses of sedative, which will reduce oxygen consumption. A sedation protocol is highly recommended. Bolus doses of a neuromuscular blocking drug may be required, particularly if using targeted temperature management (TTM) *(see below)*. Limited evidence shows that short-term infusion (≤ 48h) of short-acting neuromuscular blocking drugs given to reduce patient-ventilator dyssynchrony and risk of barotrauma in ARDS patients is not associated with an increased risk of ICU-acquired weakness and may improve outcome in these patients. Continuous electroencephalography (EEG) is recommended to detect seizures in these patients, especially when neuromuscular blockade is used. Obtain a chest radiograph to check the position of the tracheal tube, gastric tube and central venous lines, assess for pulmonary oedema, and detect complications from CPR such as a pneumothorax associated with rib fractures.

2.2. Circulation

2.2.1. Coronary reperfusion

Acute coronary syndrome (ACS) is a frequent cause of out-of-hospital cardiac arrest (OHCA): in a recent meta-analysis, the prevalence of an acute coronary artery lesion ranged from 59 % to 71 % in OHCA patients without an obvious non-cardiac aetiology. A recent consensus statement from the European Association for Percutaneous Cardiovascular Interventions (EAPCI) has emphasised that in OHCA patients, cardiac catheterisation should be performed immediately in the presence of ST-elevation and considered as soon as possible (less than two hours) in other patients in the absence of an obvious non-coronary cause, particularly if they are haemodynamically unstable. Currently, this approach in patients without STE remains controversial and is not accepted by all experts. However, it is reasonable to discuss and consider emergent cardiac catheterisation laboratory evaluation after ROSC in patients with the highest risk of a coronary cause for their cardiac arrest. Factors such as patient age, duration of CPR, haemodynamic instability, presenting cardiac rhythm, neurological status upon hospital arrival, and perceived likelihood of cardiac aetiology can influence the decision to undertake the intervention in the acute phase or to delay it until later on in the hospital stay.

2.2.2. Haemodynamic management

Post-resuscitation myocardial dysfunction causes haemodynamic instability, which manifests as hypotension, low cardiac index and arrhythmias. Perform early echocardiography in all patients in order to detect and quantify the degree of myocardial dysfunction. Post-resuscitation myocardial dysfunction often requires inotropic support, at least transiently. Based on experimental data, dobutamine is the most established treatment in this setting, but the systematic inflammatory response that occurs frequently in post-cardiac arrest patients may also cause vasoplegia and severe vasodilation. Thus, noradrenaline, with or without dobutamine, and fluid is usually the most effective

treatment. Infusion of relatively large volumes of fluid is tolerated remarkably well by patients with post-cardiac arrest syndrome. If treatment with fluid resuscitation, inotropes and vasoactive drugs is insufficient to support the circulation, consider insertion of a mechanical circulatory assistance device (e.g. IMPELLA, Abiomed, USA). Treatment may be guided by blood pressure, heart rate, urine output, rate of plasma lactate clearance, and central venous oxygen saturation. Serial echocardiography may also be used, especially in haemodynamically unstable patients. In the ICU an arterial line for continuous blood pressure monitoring is essential. Cardiac output monitoring may help to guide treatment in haemodynamically unstable patients but there is no evidence that its use affects outcome. Some centres still advocate use of an intra aortic ballon pump (IABP) in patients with cardiogenic shock, although the IABP-SHOCK II Trial failed to show that use of the IABP improved 30-day mortality in patients with myocardial infarction and cardiogenic shock. Immediately after a cardiac arrest there is typically a period of hyperkalaemia. Subsequent endogenous catecholamine release and correction of metabolic and respiratory acidosis promotes intracellular transportation of potassium, causing hypokalaemia. Hypokalaemia may predispose to ventricular arrhythmias. Give potassium to maintain the serum potassium concentration between 4.0 and 4.5 mmol l^{-1}

2.2.3. Implantable cardioverter defibrillators

Insertion of an implantable cardioverter defibrillator (ICD) should be considered in ischaemic patients with significant left ventricular dysfunction, who have been resuscitated from a ventricular arrhythmia that occurred later than 24-48 hours after a primary coronary event. ICDs may also reduce mortality in cardiac arrest survivors at risk of sudden death from structural heart diseases or inherited cardiomyopathies. In all cases, a specialised electrophysiological evaluation should be performed before discharge for placement of an ICD for secondary prevention of sudden cardiac death.

2.3. Disability and exposure

Although cardiac arrest is frequently caused by primary cardiac disease, other precipitating conditions must be excluded, particularly in hospital patients (e.g. massive blood loss, respiratory failure, pulmonary embolism). Assess the other body systems rapidly so that further resuscitation can be targeted at the patient's needs. To examine the patient properly full exposure of the body may be necessary.

Although it may not be of immediate significance to the patient's management, assess neurological function rapidly and record the Glasgow Coma Scale score *(table 13.1)*. The maximum score possible is 15; the minimum score possible is 3.

Table 13.1
The Glasgow Coma Scale score

Glasgow Coma Scale score		
Eye opening	Spontaneously	4
	To speech	3
	To pain	2
	Nil	1
Verbal	Orientated	5
	Confused	4
	Inappropriate words	3
	Incomprehensible sounds	2
	Nil	1
Best motor response	Obeys commands	6
	Localises	5
	Normal flexion	4
	Abnormal flexion	3
	Extension	2
	Nil	1

2.3. Cerebral perfusion

In many patients, autoregulation of cerebral blood flow is impaired (absent or right-shifted) for some time after cardiac arrest, which means that cerebral perfusion varies with cerebral perfusion pressure instead of being linked to neuronal activity - maintain mean arterial pressure near the patient's normal level.

2.5. Sedation

Although it has been common practice to sedate and ventilate patients for at least 24 hours after ROSC, there are no high-level data to support a defined period of ventilation, sedation and neuromuscular blockade after cardiac arrest. Patients need to be sedated adequately during treatment with TTM, and the duration of sedation and ventilation is therefore influenced by this treatment. There are no data to indicate whether or not the choice of sedation influences outcome, but a combination of opioids and hypnotics is usually used. Short-acting drugs (e.g., propofol, alfentanil, remifentanil) will enable more reliable and earlier neurological assessment and prognostication *(see prognostication below)*. Adequate sedation will reduce oxygen consumption. During hypothermia, optimal sedation can reduce or prevent shivering, which enables the target temperature to be achieved more rapidly. Use of published sedation scales for monitoring these patients (e.g. the Richmond or Ramsay Scales) may be helpful.

2.6. Control of seizures

Seizures are common after cardiac arrest and occur in approximately one-third of patients who remain comatose after ROSC. Myoclonus is most common and occurs in 18-25 %, the remainder having focal or generalized tonic-clonic seizures or a combination of seizure types. Clinical seizures, including myoclonus may or may not be of epileptic origin. Other motor manifestations could be mistaken for seizures and there are several types of myoclonus the majority being non-epileptic. Use intermittent electroencephalography (EEG) to detect epileptic activity in patients with clinical seizure manifestations. Consider continuous EEG to monitor patients with a diagnosed status epilepticus and effects of treatment. Seizures may increase the cerebral metabolic rate and have the potential to exacerbate brain injury caused by cardiac arrest: treat with sodium valproate, levetiracetam, phenytoin, benzodiazepines, propofol, or a barbiturate. Myoclonus can be particularly difficult to treat; phenytoin is often ineffective. Propofol is effective to suppress post-anoxic myoclonus. Clonazepam, sodium valproate and levetiracetam are antimyoclonic drugs that may be effective in post-anoxic myoclonus. After the first event, start maintenance therapy once potential precipitating causes (e.g. intracranial haemorrhage, electrolyte imbalance) are excluded.

Routine seizure prophylaxis in post-cardiac arrest patients is not recommended because of the risk of adverse effects and the poor response to anti-epileptic agents among patients with clinical and electrographic seizures.

2.7. Glucose control

Maintain the blood glucose at ≤ 10 mmol l^{-1} (180 mg dl^{-1}) and avoid hypoglycaemia. Do not implement strict glucose control in adult patients with ROSC after cardiac arrest because it increases the risk of hypoglycaemia.

2.8. Temperature control

Treat hyperthermia occurring after cardiac arrest with antipyretics and consider active cooling in unconscious patients.

Animal and human data indicate that mild induced hypothermia is neuroprotective and improves outcome after a period of global cerebral hypoxia-ischaemia. Cooling suppresses many of the pathways leading to delayed cell death, including apoptosis (programmed cell death). Hypothermia decreases the cerebral metabolic rate for oxygen (CMRO2) by about 6 % for each 1°C reduction in core temperature and this may reduce the release of excitatory amino acids and free radicals. Hypothermia blocks the intracellular consequences of excitotoxin exposure (high calcium and glutamate concentrations) and reduces the inflammatory response associated with the post-cardiac arrest syndrome. The term targeted temperature management (TTM) or temperature control is now preferred over the previous term therapeutic hypothermia.

- Maintain a constant, target temperature between 32°C and 36°C for those patients in whom temperature control is used (strong recommendation, moderate-quality evidence).

- TTM is recommended for all adults after CA with any initial rhythm who remain unresponsive after ROSC

- TTM is suggested for adults after OHCA with an initial nonshockable rhythm who remain unresponsive after ROSC.

- TTM is suggested for adults after IHCA with any initial rhythm who remain unresponsive after ROSC.

- If targeted temperature management is used, it is suggested that the duration is at least 24 h.

Methods of inducing and/or maintaining TTM include:

- Simple ice packs and/or wet towels are inexpensive; however, these methods may be more time consuming for nursing staff, may result in greater temperature fluctuations, and do not enable controlled rewarming. Ice cold fluids alone cannot be used to maintain hypothermia, but even the addition of simple ice packs may control the temperature adequately.

- cooling blankets or pads

- water or air circulating blankets

- water circulating gel-coated pads

- Transnasal evaporative cooling – this technique enables cooling before ROSC and is undergoing further investigation in a large multicentre randomised controlled trial.

- intravascular heat exchanger, placed usually in the femoral or subclavian veins

- Extracorporeal circulation (e.g. cardiopulmonary bypass, ECMO)

- Rebound hyperthermia is associated with worse neurological outcome. Thus, rewarming should be achieved slowly: the optimal rate is not known, but the consensus is currently about 0.25-0.5º C of rewarming per hour.

- Contraindications to targeted temperature management include: severe systemic infection and pre-existing medical coagulopathy (fibrinolytic therapy is not a contraindication to mild induced hypothermia).

2.9. Further Assessment

2.9.1. History

Obtain a comprehensive history as quickly as possible. Those involved in caring for the patient immediately before the cardiac arrest may be able to help (e.g. emergency medical personnel, general practitioner, and relatives). Specifically, symptoms of cardiac disease should be sought. Consider other causes of cardiac arrest if there is little to suggest primary cardiac disease (e.g. drug overdose, subarachnoid haemorrhage). Make a note of any delay before the start of resuscitation, and the duration of the resuscitation; this may have prognostic significance, although it is generally unreliable and certainly should not be used alone to predict outcome. The patient's baseline physiological reserve (before

the cardiac arrest) is one of the most important factors taken into consideration by the ICU team when determining whether prolonged multiple organ support is appropriate.

2.9.2. Monitoring

Continuous monitoring of ECG, arterial and possibly central venous blood pressures, cardiac output, respiratory rate, pulse oximetry, capnography, core temperature and urinary output is essential to detect changes during the period of instability that follows resuscitation from cardiac arrest. Monitor continuously the effects of medical interventions (e.g. assisted ventilation, diuretic therapy).

2.9.3. Investigations

Several physiological variables may be abnormal immediately after a cardiac arrest and urgent biochemical and cardiological investigations should be undertaken *(table 13.2)*.

- **Arterial blood gases**
Guidance on the interpretation of arterial blood gas values is given in chapter 15.

Hypoperfusion during the period of cardiac arrest will usually cause a metabolic acidosis. This will cause a low pH (acidaemia), low standard bicarbonate and a base deficit. The rate at which the acidaemia resolves in the post-resuscitation period is an important guide to the adequacy of tissue perfusion. The most effective way of correcting any acidaemia is by addressing the underlying cause. For example, poor peripheral perfusion is treated best by giving fluid and inotropic drugs and not by giving sodium bicarbonate.

The normal physiological response to a metabolic acidosis is to reduce the $PaCO_2$ by an increase in ventilation (respiratory compensation). The patient who is breathing spontaneously may fail to achieve this if ventilation is depressed by sedatives, a reduced conscious level, or significant pulmonary disease. In these cases, the $PaCO_2$ may increase, causing a combined respiratory and metabolic acidosis and profound acidaemia.

Giving bicarbonate may, paradoxically, increase intracellular acidosis, as it is converted to CO_2 with the release of hydrogen ions within the cell. Indications for bicarbonate include cardiac arrest associated with hyperkalaemia or tricyclic overdose. Do not give bicarbonate routinely to correct acidaemia after cardiac arrest.

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Table 13.2
Investigations after restoration of circulation

Full blood count
To exclude anaemia as contributor to myocardial ischaemia and provide baseline values

Biochemistry
To assess renal function
To assess electrolyte concentrations (K^+, Mg^{2+} and Ca^{2+})*
To ensure normoglycaemia
To commence serial cardiac troponin measurements
To provide baseline values

12-lead ECG
To record cardiac rhythm**
To look for evidence of acute coronary syndrome
To look for evidence of old myocardial infarction
To detect and monitor abnormalities (e.g. QT prolongation)
To provide a baseline record

Chest radiograph
To establish the position of a tracheal tube, a gastric tube, and/or a central venous catheter
To check for evidence of pulmonary oedema
To check for evidence of pulmonary aspiration
To exclude pneumothorax
To detect unintended CPR sequelae (e.g. sternal, rib fracture)
To assess cardiac contour (accurate assessment of heart size requires standard PA erect radiograph – not always practicable in the post-resuscitation situation)

Arterial blood gases
To ensure adequacy of ventilation and oxygenation
To ensure correction of acid/base imbalance

Echocardiography
To identify contributing causes to cardiac arrest.
To assess size/function of cardiac structures (chambers, valves), presence of pericardial effusion Cranial Computed tomography
If the immediate cause of cardiorespiratory arrest is not obvious
To identify causes to cardiac arrest (subarachnoid/subdural haemorrhage, intracerebral bleeding, tumour)
To identify cardiac arrest associated changes (e.g. oedema)

*   Immediately after a cardiac arrest there is typically a period of hyperkalaemia. However endogenous catecholamine release promotes influx of potassium into cells and may cause hypokalaemia. Hypokalaemia may cause ventricular arrhythmias. Give potassium to maintain the serum potassium between 4.0-4.5 mmol l^{-1}.

**   Normal sinus rhythm is required for optimal cardiac function. Atrial contraction contributes significantly to ventricular filling, especially in the presence of myocardial disease and valve disease. Loss of the sequential atrial and ventricular contraction of sinus rhythm may reduce cardiac output substantially in some patients.

2.10. Patient transfer

Following the period of initial post-resuscitation care and stabilisation, the patient will need to be transferred to an appropriate critical care environment (e.g. ICU or CCU). The decision to transfer a patient from the place where stabilisation has been achieved should be made only after discussion with senior members of the admitting team. Continue all established monitoring during the transfer and secure all cannulae, catheters, tubes and drains. Make a full re-assessment immediately before the patient is transferred using the ABCDE approach. Ensure that portable suction apparatus, an oxygen supply and a defibrillator/monitor accompany the patient and transfer team.

The transfer team should comprise individuals capable of monitoring the patient and responding appropriately to any change in patient condition, including a further cardiac arrest.

3.   Prognostication

Two thirds of those dying after admission to ICU following out-of-hospital cardiac arrest die from neurological injury. A quarter of those dying after admission to ICU following in-hospital cardiac arrest die from neurological injury. A means of predicting neurological outcome that can be applied to individual patients immediately after ROSC is required. Many studies have focused on prediction of poor long term outcome (severe cerebral disability or death), based on clinical or test findings that indicate irreversible brain injury, to enable clinicians to limit care or withdraw organ support. The implications of these prognostic tests are such that they should have 100 % specificity or zero false positive rate, i.e. no individuals should have a 'good' long-term outcome if predicted to have a poor outcome.

3.1.   Clinical examination

There are no clinical neurological signs that predict reliably poor outcome (severe cerebral disability or death) less than 24 h after cardiac arrest. Bilateral absence of pupillary light reflex at 72 h from ROSC predicts poor outcome with close to 0 % False Positive Rate (FPR) both in TTM-treated and in non-TTM-treated patients (FPR 1 [0-3] and 0 [0-8], respectively) and a relatively low sensitivity (19 % and 18 % respectively). Similar performance has been documented for bilaterally absent corneal reflex. Absence of vestibulo-ocular reflexes at ≥ 24 h and a GCS motor score of 2 or less (extension or no response to pain) at ≥ 72 h are less reliable. Other clinical signs, including myoclonus, are not recommended for predicting

poor outcome. The presence of myoclonic status in adults is strongly associated with poor outcome but rare cases of good neurological recovery from this situation have been described and accurate diagnosis of myoclonic status is problematic.

3.2. Electrophysiology

3.2.1. Short-latency somatosensory evoked potentials (SSEPs)

In most prognostication studies bilateral absence of N20 SSEP has been used as a criterion for deciding on withdrawal of life-sustaining treatment (WLST), with a consequent risk of self-fulfilling prophecy. SSEP results are more likely to influence physicians' and families' WLST decisions than those of clinical examination or EEG.

3.2.2. Electroencephalography

In TTM-treated patients, absence of EEG background reactivity predicts poor outcome with at 48 h-72 h from ROSC. Most of the prognostication studies on absent EEG reactivity after cardiac arrest are from the same group of investigators. Limitations of EEG reactivity include lack of standardisation as concerns the stimulation modality and modest interrater agreement. Apart from its prognostic significance, recording of EEG – either continuous or intermittent – in comatose survivors of cardiac arrest both during TTM and after rewarming is helpful to assess the level of consciousness – which may be masked by prolonged sedation, neuromuscular dysfunction or myoclonus – and to detect and treat non-convulsive seizures which may occur in about one quarter of comatose survivors of cardiac arrest.

3.3. Biomarkers

NSE and S-100B are protein biomarkers that are released following injury to neurons and glial cells, respectively. Their blood values after cardiac arrest are likely to correlate with the extent of anoxic-ischaemic neurological injury and, therefore, with the severity of neurological outcome. S-100B is less well documented than is NSE. Advantages of biomarkers over both EEG and clinical examination include quantitative results and likely independence from the effects of sedatives. Their main limitation as prognosticators is that it is difficult to find a consistent threshold for identifying patients destined to a poor outcome with a high degree of certainty. In fact, serum concentrations of biomarkers are per se continuous variables, which limits their applicability for predicting a dichotomous outcome,

3.4. Imaging

Many imaging modalities (magnetic resonance imaging [MRI], computed tomography [CT], single photon emission computed tomography [SPECT], cerebral angiography, transcranial Doppler, nuclear medicine, near infra-red spectroscopy [NIRS]) have been studied to determine their utility for prediction of outcome in adult survivors of cardiac arrest. Based on the available evidence, none of these imaging modalities will predict reliably outcome of comatose cardiac arrest survivors.
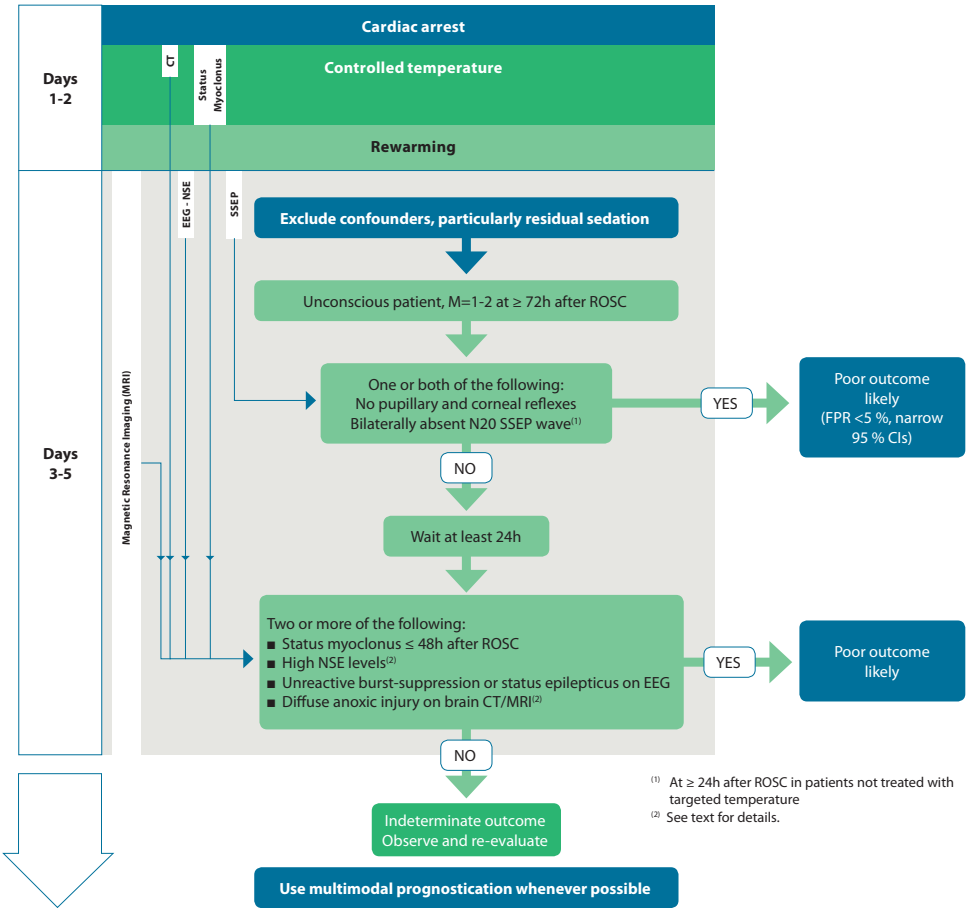
3.5.   Suggested prognostication strategy

A careful clinical neurological examination remains the foundation for prognostication of the comatose patient after cardiac arrest. Perform a thorough clinical examination daily to detect signs of neurological recovery such as purposeful movements or to identify a clinical picture suggesting that brain death has occurred.

The process of brain recovery following global post-anoxic injury is completed within 72 h from arrest in most patients. However, in patients who have received sedatives ≤ 12 h before the 72 h post ROSC neurological assessment, the reliability of clinical examination may be reduced. Before decisive assessment is performed, major confounders must be excluded; apart from sedation and neuromuscular blockade, these include hypothermia, severe hypotension, hypoglycaemia, and metabolic and respiratory derangements. Suspend sedatives and neuromuscular blocking drugs for long enough to avoid interference with clinical examination. Short-acting drugs are preferred whenever possible. When residual sedation/paralysis is suspected, consider using antidotes to reverse the effects of these drugs.

The prognostication strategy algorithm *(figure 13.2)* is applicable to all patients who remain comatose with an absent or extensor motor response to pain at ≥ 72 h from ROSC. Results of earlier prognostic tests are also considered at this time point.

Evaluate the most robust predictors first. They include bilaterally absent pupillary reflexes at ≥ 72 h from ROSC and bilaterally absent SSEP N20 wave after rewarming (this last sign can be evaluated at ≥ 24 h from ROSC in patients who have not been treated with controlled temperature). Based on expert opinion, we suggest combining the absence of pupillary reflexes with those of corneal reflexes for predicting poor outcome at this time point. Ocular reflexes and SSEPs maintain their predictive value irrespective of target temperature. If none of the signs above is present to predict a poor outcome, a group of less accurate predictors can be evaluated, but the degree of confidence in their prediction will be lower. These predictors include the presence of early status myoclonus (within 48 h from ROSC), high values of serum neuron specific enolase(NSE) at 48 h-72 h after ROSC, an unreactive malignant EEG pattern (burst-suppression, status epilepticus) after rewarming, the presence of a marked reduction of the grey/white matter (GM/WM) ratio or sulcal effacement on brain CT within 24 h after ROSC or the presence of diffuse ischaemic changes on brain MRI at 2-5 days after ROSC. Based on expert opinion, we suggest waiting at least 24 h after the first prognostication assessment and confirming unconsciousness with a Glasgow motor score of 1-2 before using this second set of predictors. We also suggest combining at least two of these predictors for prognostication.

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Figure 13.2
Prognostication strategy

Cardiac arrest

Days 1-2

CT

Status Myoclonus

Controlled temperature

Rewarming

EEG · NSE

SSEP

Exclude confounders, particularly residual sedation

Unconscious patient, M=1-2 at ≥ 72h after ROSC

Days 3-5

Magnetic Resonance Imaging (MRI)

One or both of the following:
No pupillary and corneal reflexes
Bilaterally absent N20 SSEP wave[1]

YES → Poor outcome likely (FPR <5 %, narrow 95 % CIs)

NO

Wait at least 24h

Two or more of the following:
■ Status myoclonus ≤ 48h after ROSC
■ High NSE levels[2]
■ Unreactive burst-suppression or status epilepticus on EEG
■ Diffuse anoxic injury on brain CT/MRI[2]

YES → Poor outcome likely

NO

[1] At ≥ 24h after ROSC in patients not treated with targeted temperature
[2] See text for details.

Indeterminate outcome
Observe and re-evaluate

Use multimodal prognostication whenever possible

4.   Organ donation

Organ donation should be considered in those who have achieved ROSC and who fulfil criteria for death using neurological criteria. In those comatose patients in whom a decision is made to withdraw life-sustaining therapy, organ donation should be considered after circulatory death occurs. Organ donation can also be considered in individuals where CPR is not successful in achieving ROSC. All decisions concerning organ donation must follow local legal and ethical requirements, as these vary in different settings.

5.   Care of the resuscitation team

Audit all resuscitation attempts and, ideally, send these data to a national cardiac arrest audit *(chapter 2)*.

Feedback for the resuscitation team should be constructive and not based on a fault/blame culture. Whether the resuscitation attempt was successful or not, the patient's relatives will require considerable support. Consider the pastoral needs of all those associated with the arrest.

6.   Cardiac arrest centres

Specialist cardiac arrest centres and systems of care may be effective. Despite the lack of high-quality data to support implementation of cardiac arrest centres, it seems likely that regionalisation of post-cardiac arrest care will be adopted in most countries.

KEY LEARNING POINTS

- After cardiac arrest, return of spontaneous circulation is just the first stage in a continuum of resuscitation.

- The quality of post-resuscitation care will influence significantly the patient's final outcome.

- These patients require appropriate monitoring, safe transfer to a critical care environment, and continued organ support.

- The post-cardiac arrest syndrome comprises post-cardiac arrest brain injury, post-cardiac arrest myocardial dysfunction, the systemic ischaemia/reperfusion response, and persistence of precipitating pathology.

- Our ability to predict the final neurological outcome for those patients remaining comatose after cardiopulmonary resuscitation remains very poor.

13

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Chapter 14.

Pre-hospital cardiac arrest

LEARNING OUTCOMES

To understand:

- the role of telephone assisted/dispatcher assisted cardiopulmonary resuscitation (CPR)
- the current position on CPR versus defibrillation first
- how to change over efficiently from an AED to a manual defibrillator
- the importance of effective handover to hospital staff
- rules for stopping resuscitation
- the potential role of cardiac arrest centres

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1.   Introduction

The aim of the pre-hospital section included in the European Resuscitation Council Advanced Life Support (ALS) course manual is to bring together resuscitation topics of specific relevance to the pre-hospital emergency medical services (EMS). The increased emphasis on the importance of minimally interrupted high-quality chest compressions and reducing the pre-shock pause by continuing chest compressions while the defibrillator is charged demands a well structured, monitored training programme for pre-hospital EMS practitioners. This should include comprehensive competency-based training and regular opportunities to refresh skills. It is recognised that in most cases pre-hospital resuscitation has to be managed by fewer practitioners than would normally be present at an in-hospital arrest; also transportation to a receiving centre adds an extra dimension. This emphasises the need for a structured and disciplined approach. The ERC ALS course provides the ideal platform to develop and practise resuscitation skills and strengthen the multidisciplinary team approach.

2.   Personnel and Interventions

There is considerable variation across Europe in the structure and process of emergency medical services (EMS) systems. Some countries have adopted almost exclusively paramedic/emergency medical technician (EMT)-based systems while other incorporate prehospital physicians to a greater or lesser extent. Although some studies have documented higher survival rates after cardiac arrest in EMS systems that include experienced physicians, compared with those that rely on non-physician providers, some other comparisons have found no difference in survival between systems using paramedics or physicians as part of the response. Well-organised non-physician systems with highly trained paramedics have also reported high survival rates. Given the inconsistent evidence, the inclusion or exclusion of physicians among prehospital personnel responding to cardiac arrests will depend largely on existing local policy.

3.   Dispatcher assisted CPR

The emergency medical dispatcher plays a critical role in the diagnosis of cardiac arrest, the provision of dispatcher assisted CPR (also known as telephone CPR), the location and dispatch of an automated external defibrillator and dispatch of a high priority EMS response. The sooner the emergency services are called, the earlier appropriate treatment can be initiated and supported. Confirmation of cardiac arrest, at the earliest opportunity is critical. If the dispatcher recognises cardiac arrest, survival is more likely because appropriate measures can be taken. Enhancing dispatcher ability to identify cardiac arrest, and optimising emergency medical dispatcher processes, may be cost-effective solutions to improve outcomes from cardiac arrest.

Use of scripted dispatch protocols within emergency medical communication centres, including specific questions to improve cardiac arrest recognition may be helpful. Patients who are **unresponsive** and **not breathing normally** should be presumed to be in cardiac arrest. Adherence to such protocols may help improve cardiac arrest recognition, whereas failure to adhere to protocols reduces rates of cardiac arrest recognition by dispatchers as well as the provision of telephone-CPR. Bystander CPR rates are low in many communities. Dispatcher-assisted CPR (telephone-CPR) instructions have been demonstrated to improve bystander CPR rates, reduce the time to first CPR, increase the number of chest compressions delivered and improve patient outcomes following out-of-hospital cardiac arrest (OHCA) in all patient groups.

Dispatchers should provide telephone-CPR instructions in all cases of suspected cardiac arrest unless a trained provider is already delivering CPR. Where instructions are required for an adult victim, dispatchers should provide chest-compression-only CPR instructions.

If the victim is a child, dispatchers should instruct callers to provide both ventilations and chest compressions. Dispatchers should therefore be trained to provide instructions for both techniques.

4.   Pre-hospital airway management

There is insufficient evidence to support or refute the use of any specific technique to maintain an airway and provide ventilation in adults with pre-hospital cardiac arrest. Tracheal intubation has been perceived as the optimal method of providing and maintaining a clear and secure airway during cardiac arrest but data are accumulating on the challenges associated with pre-hospital intubation. It is now strongly recommended that tracheal intubation should be used only when trained personnel are available to carry out the procedure with a high level of skill and confidence. In the absence of experienced personnel the use of supraglottic airway devices (SADs) during CPR is probably more rational.

However, there are only poor-quality data on the pre-hospital use of these devices during cardiac arrest as the teams working in the EMS are structured in different ways (physician staffed, ALS units, BLS units, rendezvous systems).

Tracheal intubation and the use of SADs is discussed in more detail in chapter 7.

4.1.   Ventilation

The majority of CPR patients in the pre-hospital setting will be ventilated following airway management. Depending on the equipment available, bag-valve devices or simple ventilators will be used. To avoid potential problems like displacement of the airway device or secondary breathing problems, monitoring should include waveform capnography where available and continuous saturation measurement. Tidal volumes of about 6-7 ml kg^{-1} ideal body weight at a rate of 10-12 min^{-1} will provide adequate oxygenation and ventilation. Hypercapnia as well as hypocapnia should be avoided as these may worsen outcome.

5.   Defibrillation

5.1.   CPR versus defibrillation first

Defibrillation is a key link in the Chain of Survival and is one of the few interventions that has been shown to improve outcome from ventricular fibrillation/pulseless ventricular tachycardia (VF/pVT) cardiac arrest. The probability of successful defibrillation and subsequent survival to hospital discharge declines rapidly with time and the ability to deliver early defibrillation is one of the most important factors in determining survival from cardiac arrest.

EMS personnel should provide high-quality CPR while a defibrillator is retrieved, applied and charged. Defibrillation should not be delayed longer than needed to establish the need for defibrillation and charging. The routine delivery of a pre-specified period of CPR (e.g. two or three minutes) before rhythm analysis and a shock is delivered is not recommended.

5.2.   Transition from AED to manual defibrillator

In many situations, an automated external defibrillator (AED) is used to provide initial

defibrillation but is subsequently swapped for a manual defibrillator on arrival of EMS personnel. If such a swap is done without considering the phase of the AED cycle, the next shock may be delayed, which may compromise outcome. For this reason, EMS personnel should leave the AED connected while securing the airway and IV access. The AED can be left attached for the next rhythm analysis and, if indicated, shock delivery, before being swapped for the manual defibrillator.

6. CPR during transportation to hospital

During transportation to hospital, manual CPR is often performed poorly; mechanical CPR can maintain high-quality CPR during transfer by land ambulance or helicopter. Automated mechanical chest compression devices may enable the delivery of high-quality compressions in a moving ambulance. Data from the US American CARES (Cardiac Arrest Registry to Enhance Survival) registry shows that 45% of participating EMS services use mechanical devices. Since Guidelines 2010 there have been three large RCTs enrolling 7582 patients that have shown no clear advantage from the routine use of automated mechanical chest compression devices for OHCA.

Automated mechanical chest compression devices should not be used routinely to replace manual chest compressions. Automated mechanical chest compression devices are a reasonable alternative to high-quality manual chest compressions in situations where sustained high-quality manual chest compressions are impractical or compromise provider safety. Interruptions to CPR during device deployment should be avoided. Healthcare personnel who use mechanical CPR should do so only within a structured, monitored programme, which should include comprehensive competency-based training and regular opportunities to refresh skills.

7. Hospital handover

If a cardiac arrest victim is transported to hospital, clear and accurate communication and documentation are essential elements of the handover to hospital staff. Vital information may be lost or misinterpreted if communication between EMS practitioners and hospital staff is not effective.

A pre-alert message should be routine and is essential in ongoing resuscitation on transport to ensure that emergency department staff and/or the hospital resuscitation team are ready to receive the patient. This gives time for the hospital resuscitation team to elect a team leader and assign roles to team members. Specific interventions to treat potentially reversible causes or specialist intervention can be arranged.

Emergency medical services personnel need to be completely focused on communicating vital information about the patient, the circumstances surrounding the resuscitation and actions taken. This has to be done against a background of considerable activity and with the added pressure of time. Hospital staff need to be focused on beginning their own assessment and treatment of the patient, but this must not prevent them from listening to the vital information provided by the EMS personnel. A structured approach will enhance the handover and make the transition as rapid and effective as possible. Communication

failure has been cited as a contributory factor in cases of error and harm to patients.

The ALS course enables pre-hospital and hospital staff to understand each other's role and develops the multidisciplinary team approach.

8.   Termination of Resuscitation Rules

The 'basic life support termination of resuscitation rule' is predictive of death when applied by defibrillation-only emergency medical technicians. The rule recommends termination when there is no ROSC, no shocks are administered and EMS personnel do not witness the arrest. Several studies have shown external generalisability of this rule. More recent studies show that EMS systems providing ALS interventions can also use this BLS rule and therefore termed it the 'universal' termination of resuscitation rule.

Additional studies have shown associations with futility of certain variables such as no ROSC at scene; non-shockable rhythm; unwitnessed arrest; no bystander CPR, call response time and patient demographics.

Termination of resuscitation rules for in-hospital cardiac arrest are less reliable although EMS rules may be useful for those with out-of-hospital cardiac arrest who have ongoing resuscitation in the emergency department.

Prospectively validated termination of resuscitation rules can be used to guide termination of prehospital CPR in adults; however, these must be validated in an EMS system similar to the one in which implementation is proposed. Termination of resuscitation rules may require integration with guidance on suitability for extracorporeal CPR (eCPR) or organ donation.

9.   Regionalisation of post resuscitation care

Several studies with historical control groups have shown improved survival after implementation of a comprehensive package of post-resuscitation care that includes therapeutic hypothermia and percutaneous coronary intervention. There is also evidence of improved survival after out-of-hospital cardiac arrest in large hospitals with cardiac catheter facilities compared with smaller hospitals with no cardiac catheter facilities.

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Several studies of out-of-hospital adult cardiac arrest failed to demonstrate any effect of transport interval from the scene to the receiving hospital on survival to hospital discharge if ROSC was achieved at the scene and transport intervals were short (3-11 min). This implies that it may be safe to bypass local hospitals and transport the post-cardiac arrest patient to a regional cardiac arrest centre. There is indirect evidence that regional cardiac resuscitation systems of care improve outcome after ST elevation myocardial infarction (STEMI).

The implication from all these data is that specialist cardiac arrest centres and systems of care may be effective but, as yet, there is no direct evidence to support this hypothesis.

KEY LEARNING POINTS

- In adults, telephone assisted/dispatcher assisted compression-only CPR produces better survival rates than telephone-advised conventional CPR.

- EMS personnel should provide high-quality CPR while preparing, applying and charging a defibrillator, but a routine, specified period of CPR before shock delivery is not recommended.

- Tracheal intubation should be attempted only by those with a high level of skill and experience with the technique.

- Waveform capnography is the most sensitive and specific method for confirming the position of a tracheal tube in victims of cardiac arrest.

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Chapter 15.

Blood gas analysis and pulse oximetry

LEARNING OUTCOMES

To understand:

- the terms used to describe the results of arterial blood gas analysis
- how respiration and metabolism are linked
- how to use the 5-step approach to analyse arterial blood gas results
- the principles of pulse oximetry
- the safe and effective use of oxygen

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1.   Introduction

Interpreting the analysis of an arterial blood sample to determine a patient's acid-base status and respiratory gas exchange is a key component in the management of any ill patient and, particularly, in the peri-arrest situation. Although there is often a great temptation to try and analyse the numerical data in isolation, it is essential to have a system to ensure that nothing is overlooked or misinterpreted; as when reading an ECG, this starts with asking "how is the patient?" This should include any known history along with details of current oxygen therapy and medications.

There are usually four key pieces of information contained in the results of analysis of an arterial blood sample:

- pH
- $PaCO_2$ (partial pressure of carbon dioxide in arterial blood)
- Bicarbonate and base excess
- PaO_2 (partial pressure of oxygen in arterial blood)

In order to interpret these results, we first need to understand what each means. Normal ranges are given in the text; however, these will vary slightly between institutions.

2.   pH

The acidity or alkalinity of the blood (or any solution) is determined by the concentration of hydrogen ions [H^+]; the greater the concentration, the more acid the solution. In the body, the concentration of hydrogen ions is extremely low, normally around 40 nanomoles per litre (nmol l^{-1}), where a nanomole is 1 billionth of a mole (a mole is the molecular weight of a substance in grams, i.e. for hydrogen it would be 2 g). To put this into perspective, sodium ions (Na^+) are present in a concentration of 135 millimoles per litre (mmol l^{-1}), i.e. 3 million times greater. In order to make dealing with such small numbers easier, we use the pH scale; this is a logarithmic scale expressing the hydrogen ion concentration between 1 and 14. The pH of a normal arterial blood sample lies between 7.35 and 7.45, or [H^+] 44 - 36 nmol l^{-1}. There are two key points to remember about the pH scale:

1. The numerical value of pH changes inversely with hydrogen ion concentration. Consequently a decrease in blood pH below 7.35 indicates an increase in [H^+] above normal, a condition referred to as an acidaemia. Conversely, an increase in blood pH above 7.45 indicates a reduction in [H^+] below normal, a condition referred to as an alkalaemia. Clinicians often use the terms acidosis and alkalosis respectively to describe these situations. Strictly speaking, these terms refer to the processes that lead to the changes in pH, and it is in this context that they will be used in this manual.

2. Small changes in pH represent big changes in hydrogen ion [H^+] concentration. For example, a pH change from 7.4 to 7.1 means that the hydrogen ion concentration has increased from 40 nmol l^{-1} to 80 nmol l^{-1}, i.e. it has doubled for a pH change of 0.3.

Many of the complex reactions within cells are controlled by enzymes that function only within a very narrow pH range; hence, normal pH is controlled tightly between 7.35 and 7.45. However, each day during normal activity we produce massive amounts of hydrogen ions (approximately 14 500 000 000 nmol), which if unchecked would cause a substantial decrease in pH (acidaemia) before they could be excreted. To prevent this happening the body has a series of substances known as buffers that take up hydrogen ions and thereby prevent the development of an acidaemia. The major intracellular buffers are proteins, phosphate and haemoglobin (within red blood cells) and the extracellular buffers are plasma proteins and bicarbonate *(see below)*.

Clearly the buffering system is only a temporary solution to the production of acids; ultimately they will all be consumed and acids will start to accumulate. A system is therefore required to eliminate the acids and thereby regenerate the buffers. This is achieved by the lungs and kidneys.

3.   Partial pressure

We normally use percentages to describe the composition of a mixture of gases, a good example being air: 21 % oxygen, 78 % nitrogen, 0.04 % carbon dioxide. However the number of molecules of a gas in a mixture is better described by referring to its partial pressure. The partial pressure is the contribution each gas in a mixture makes to the total pressure. The

importance of using this measure is best demonstrated by the fact that if we double the total pressure of a mixture, the partial pressures of the constituents are doubled, but the percentages remain the same. We breathe gases at atmospheric pressure or 1 atmosphere, very close to a pressure of 100 kiloPascals (kPa) or 750 mmHg (1 kPa = 7.5 mmHg). As a result, when breathing air, the contribution (partial pressure) of nitrogen is 78 % of 100 kPa or 78 kPa and oxygen 21 % of 100 kPa or 21 kPa. When breathing 40 % oxygen, the partial pressure of the oxygen in the inspired gas is 40 kPa.

At atmospheric pressure, the partial pressure of a gas in a mixture (in kPa) is numerically the same as the percentage (%) of the gas by volume.

When a gas is dissolved in a liquid (e.g. blood) the partial pressure within the liquid is the same as in the gas in contact with the liquid. This enables us to measure the partial pressure of oxygen and carbon dioxide in blood.

In summary, the partial pressure of a gas is a measure of the concentration of the gas in the medium it is in and is expressed as PmediumGas, e.g. $PaCO_2$ is the partial pressure (P) of carbon dioxide (CO_2) in arterial blood (a).

4. $PaCO_2$

Carbon dioxide (CO_2) is an important waste product of metabolism. Under normal circumstances, it is transported in the blood to the lungs where it is excreted during expiration. For transport to the lungs, it is either combined with protein or haemoglobin, or is dissolved in plasma where it reacts with water to form hydrogen ions and bicarbonate (HCO_3):

$$CO_2 + H_2O = H^+ + HCO_3^-$$
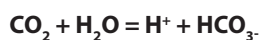
The normal $PaCO_2$ is 5.3 kPa with a range of 4.7-6.0 kPa.

In the lungs, the reaction proceeds in reverse: CO_2 is generated and expired. From this reaction, we can see that CO_2 behaves as an acid: any increase in $PaCO_2$ will cause the reaction to move to the right and increase the hydrogen ion concentration with the subsequent development of an acidaemia. There will, of course, be the same increase in bicarbonate concentration but, as this is only nanomoles, it has little effect on the overall total concentration of 22-26 mmol l^{-1}. If the metabolic production of CO_2 is constant, the only factor that affects the amount in the blood is the rate at which it is removed by alveolar ventilation. A decrease in alveolar ventilation will reduce excretion of CO_2 causing an increase in $PaCO_2$ and the production of more hydrogen ions. If the pH decreases below 7.35 an acidaemia has been produced. As the primary cause of the acidaemia is a problem with the respiratory system, we call this process a respiratory acidosis.

Conversely, an increase in alveolar ventilation that removes CO_2 faster than it is generated reduces $PaCO_2$ and moves the reaction to the left, reducing the concentration of hydrogen ions. As a result the pH will increase and if it exceeds 7.45 an alkalaemia has been produced. Again, the primary cause is the respiratory system and we call this process a respiratory alkalosis.

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It is easy to understand therefore how even brief periods of apnoea, as occurs during cardiac arrest, result in a respiratory acidosis. However, under normal circumstances, the respiratory centre in the brain stem is very sensitive to blood [H^+] and within a few minutes rapidly increases alveolar ventilation. This increases CO_2 excretion, reduces $PaCO_2$, decreases [H^+] and returns pH to normal.

The lungs are the primary mechanism by which [H^+] is adjusted by regulating $PaCO_2$.

5.   Bicarbonate and base excess

5.1.   Bicarbonate

Bicarbonate (HCO_3.) is the most important buffer. It is generated by the kidneys and is measured easily in an arterial blood sample. It can be thought of as the opposite of an acid and as such is also called a base. When bicarbonate buffers hydrogen ions, carbon dioxide and water are produced, and it is via this route that the vast majority of acids (90 %) are excreted each day. However, the acids not eliminated by the respiratory system can also be buffered as shown below. The reaction below moves to the right and bicarbonate neutralises the effect of the H^+ and prevents a decrease in plasma pH. In the kidneys, the reaction proceeds to the left, the H^+ is excreted in the urine and bicarbonate filtered and returned to the plasma. Depending on the acid load, the kidneys will excrete either acid or alkaline urine.

$H^+ + HCO_3. = H_2CO_3.$
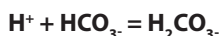
Under normal circumstances, the concentration of bicarbonate is 22-26 mmol l^{-1}.

If there is an acute increase in the acid load, although the respiratory system will try and increase excretion of carbon dioxide, bicarbonate will decrease as it buffers the extra H^+. Once the reserves of bicarbonate are used, H^+ will accumulate decreasing the pH. Unlike the respiratory system, the kidneys respond slowly and it takes several days for additional bicarbonate to be produced to meet the demand to buffer the extra acid. If the kidneys fail to produce sufficient bicarbonate the resultant metabolic acidosis will lead to a decrease in pH below 7.35 (acidaemia).

Occasionally, there is an excess of bicarbonate. This will have the effect of excessive buffering of hydrogen ions and will produce a metabolic alkalosis and increase the pH above 7.45 (alkalaemia).

5.2.   Base excess

This is a measure of the amount of excess acid or base in the blood as a result of a metabolic derangement. It is calculated as the amount of strong acid or base that would have to be added to a blood sample with an abnormal pH to restore it to normal (pH 7.4). Consequently, a patient with a base excess of 8 mmol l^{-1} would require 8 mmol l-1 of strong acid to return their pH to normal; that is they have a metabolic alkalosis (compare with bicarbonate which

would be raised, so both move in the same direction). Conversely, a patient with a base deficit of 8 mmol l^{-1} will require the addition of 8 mmol l^{-1} of strong base to normalise their pH (again, compare with bicarbonate which would be reduced). Unfortunately, the term "negative base excess" is used instead of base deficit and in the example above, the patient would have a negative base excess of - 8 mmol l^{-1}.

As a result, the normal values of base excess are + 2 to - 2 mmol l^{-1}.

A base excess more negative than - 2 mmol l^{-1} indicates a metabolic acidosis.

A base excess greater than + 2 mmol l^{-1} indicates a metabolic alkalosis.

6.   The respiratory - metabolic link

From the above we can see that the body has two systems for ensuring a stable internal environment and preventing the development of an acidosis. Additional protection is provided by the fact that the two systems are linked and can compensate for derangements in each other. This link is provided by the presence of carbonic acid (H_2CO_3), which is dependent on the presence of an enzyme called carbonic anhydrase, present in both red blood cells and the kidneys, and ideally situated to facilitate the link between the two systems.

$$CO_2 + H_2O \rightleftharpoons H_2CO_3 \rightleftharpoons H^+ + HCO_3^-$$

Although this link exists, the ability of each system to compensate for the other is not instantaneous, but becomes more marked when the initial disturbance in one system is prolonged. A typical example demonstrating the link between the two systems is a patient with chronic obstructive pulmonary disease (COPD). This condition results in diminished capacity to excrete carbon dioxide and a respiratory acidosis. If left uncompensated, this would result in a persistent acidaemia, but the increase in carbon dioxide drives the reaction above to the right, with the production of carbonic acid (H_2CO_3). In the kidneys this has the effect of increasing H^+ ions which are excreted in the urine while at the same time increasing bicarbonate production to buffer the H^+ ions in the plasma. As a result the patient has a respiratory acidosis (increased $PaCO_2$) with a compensatory metabolic alkalosis (increased bicarbonate) and the pH will return close to normal.

A different example is a diabetic in ketoacidosis (strictly speaking ketoacidaemia). When the excess ketoacids exceed the kidney's ability for excretion, they are buffered, which consumes plasma bicarbonate. Increasing bicarbonate production takes several days. However, the reaction above can also move to the left by increasing ventilation and reducing $PaCO_2$; in effect, converting the H^+ to CO_2. Consequently, the patient has a metabolic acidosis (reduced bicarbonate) with a compensatory respiratory alkalosis (reduced $PaCO_2$).

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7. PaO$_2$

The concentration of oxygen in inspired air is 21 % - representing a partial pressure of 21 kPa. This is gradually reduced as the air passes down the respiratory tract, firstly because of the addition of water vapour and, in the alveoli, by the addition of carbon dioxide so that here it is normally around 13 kPa. However, the partial pressure of oxygen in arterial blood (PaO$_2$) is always lower than alveolar; the extent of this gradient is determined largely by the presence of any lung disease. In a healthy individual breathing air, the PaO$_2$ is normally higher than 11 kPa i.e. about 10 kPa lower than the inspired partial pressure. This can be used as a rule of thumb to estimate the PaO$_2$ for any given inspired concentration, in that it should be numerically about 10 less than the inspired concentration (%). For example, 40 % inspired oxygen should result in a PaO$_2$ of approximately 30 kPa. With increasing lung injury, the gap between inspired concentration and PaO$_2$ increases. This is important to recognise because for someone breathing 50 % oxygen a PaO$_2$ of 13 kPa is not 'normal'.

Interestingly, PaO$_2$ also decreases slightly with age, reaching 10 kPa at around 75 years, but then climbs again and plateaus at around 11 kPa at 85 years.

8. Interpreting the results

Interpretation of the result of blood gas analysis is achieved best by following strictly five steps. For clarity, only changes in base excess are discussed; however, bicarbonate will also change numerically in the same direction.

8.1. Step 1

How is the patient?
This will often provide useful clues to help with interpretation of the results. For example, one might reasonably predict that analysis of arterial blood shortly after successful resuscitation would show signs of a respiratory acidosis caused by a period of inadequate ventilation and a metabolic acidosis due to the period of anaerobic respiration during the arrest producing lactic acid. Consequently, we would expect the patient to have a very low pH with changes in both PaCO$_2$ and base excess. A patient with a well-compensated, chronic condition will usually display clues about the primary cause and secondary compensation. Without the clinical history, the results of a blood gas analysis from a patient with COPD could be misinterpreted as a primary metabolic alkalosis with a compensatory respiratory acidosis.

8.2. Step 2

Is the patient hypoxaemic?
The PaO$_2$ while breathing room air should be 10.0-13.0 kPa. However, if the patient is receiving supplemental oxygen, the PaO$_2$ must be interpreted in light of the inspired oxygen concentration. As discussed above, the inspired partial pressure of oxygen can be regarded as the numerical equivalent of the inspired concentration (%). If there is a difference of greater than 10 between the two values, there is a defect in oxygenation, proportional to the magnitude of the difference.

8.3. Step 3

Is the patient acidaemic (pH < 7.35) or alkalaemic (pH> 7.45)?

If the pH is within or very close to the normal range then this suggests normality or a chronic condition with full compensation. In principle, the body never overcompensates and this should enable the primary problem to be determined. If necessary, seek more clinical information about the patient.

8.4. Step 4

What has happened to the $PaCO_2$?

In other words, is the abnormality wholly or partially due to a defect in the respiratory system?

If the pH is < 7.35 (acidaemia):

4a. Is the $PaCO_2$ increased (> 6.0 kPa)?

    If so, there is a **respiratory acidosis** that may be accounting for all or part of the derangement. There could also be a metabolic component, see step 5a.

If the pH is > 7.45 (alkalaemia):

4b. Is the $PaCO_2$ reduced (< 4.7 kPa)?

    If so, there is a **respiratory alkalosis**, but this is an unusual isolated finding in a patient breathing spontaneously, with a normal respiratory rate. It is seen more often in patients who are being mechanically ventilated with excessively high rates and/or tidal volumes. As a result, $PaCO_2$ decreases, there is a reduction in H^+ and an alkalosis develops.

8.5. Step 5

What has happened to the base excess or bicarbonate?

In other words, is the abnormality wholly or partially due to a defect in the metabolic system?

If the pH is < 7.35 (acidaemia):

5a. Is the base excess reduced (more negative than minus 2 mmol l^{-1}), and /or the bicarbonate reduced (< 22 mmol l^{-1})? If so, there is a metabolic acidosis accounting for all or part of the derangement. There could be a respiratory component if the $PaCO_2$ is also increased - see step 4a, a situation commonly seen after a cardiac arrest.

If the pH is > 7.45 (alkalaemia):

5b. Is the base excess increased (> + 2 mmol l^{-1}) and/or the bicarbonate increased (> 26 mmol l^{-1})? If so, there is a metabolic alkalosis accounting for all or part of the derangement. There could be a respiratory component if the $PaCO_2$ is also decreased - see step 4b, but this would be very unusual.

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8.6.   Derangements of both PaCO$_2$ and base excess or bicarbonate – compensation

The results of ABGs may show changes in both the respiratory and metabolic components, but with minimal disturbance of the pH. This is the result of compensation; both the respiratory and metabolic systems are capable of reacting to changes in the other, the aim being to minimise long term changes in pH. Four examples follow:

8.6.1. Example 1

pH < 7.40, with a increased PaCO$_2$ (> 6.0 kPa) and increased base excess (> + 2 mmol l^{-1}) or bicarbonate (> 26 mmol l^{-1}).

The tendency towards an acidaemia indicates that this is the primary problem and the increased PaCO$_2$ indicates that it is a **respiratory acidosis**. The increased base excess/bicarbonate represents a **compensatory metabolic alkalosis**, bringing the pH back towards normality.

8.6.2. Example 2

pH < 7.40, with a decreased base excess (< - 2 mmol l^{-1}) or bicarbonate (< 22 mmol l^{-1}) and decreased PaCO$_2$ (< 4.7 kPa).

The tendency towards an acidaemia indicates that this is the primary problem and the decreased base excess/bicarbonate suggests that it is a **metabolic acidosis**. The decrease in PaCO$_2$ represents a **compensatory respiratory alkalosis**, bringing the pH back towards normality.

8.6.3. Example 3

pH > 7.40, with increased base excess (> + 2 mmol l^{-1}) or bicarbonate (> 26 mmol l^{-1}) and increased PaCO$_2$ (> 6.0 kPa).

The tendency towards an alkalaemia indicates that this is the primary problem and the increase in base excess/bicarbonate suggests that it is primarily a **metabolic alkalosis**. The increased PaCO$_2$ is **respiratory compensation** bringing the pH back towards normality. This picture may be seen where there is loss of acid from the body e.g. prolonged vomiting of gastric contents (hydrochloric acid) and also occurs in chronic hypokalaemia. In this case, the body compensates by moving potassium from intracellular to extracellular in exchange for hydrogen ions. The pH increases and CO$_2$ is retained to try and compensate.

8.6.4. Example 4

pH > 7.40, with a decreased PaCO$_2$ (< 4.7 kPa) and decreased base excess (< - 2 mmol l^{-1}) or bicarbonate (< 22 mmol l^{-1}).

The tendency towards an alkalaemia indicates that this is the primary problem and the decrease in $PaCO_2$ **suggests that this is primarily a** respiratory alkalosis. The decrease in base excess/bicarbonate is the **metabolic compensation** bringing the pH back towards normality. This is not a common finding, but may be seen after a few days when hyperventilation is used to help control intracranial pressure in patients with brain injury.

Using the above, work through cases 4 and 5 at the end of this chapter.

There is one final situation that deserves mention and is important to identify: an ill patient with a pH < 7.4, a normal $PaCO_2$ (4-6.0 kPa) and a decreased base excess (< - 2 mmol l^{-1}) or bicarbonate (< 22 mmol l^{-1}).

This is most likely to represent the situation of a metabolic acidosis in a patient with chronic carbon dioxide retention. The patient is trying to compensate by lowering their carbon dioxide (to cause a compensatory respiratory alkalosis), but they are starting from a higher $PaCO_2$. Their lung disease will limit the amount of CO_2 they can excrete, thereby preventing it decreasing any further. Once again it illustrates the importance of having information about the patient as identified at the beginning.

9. Practical aspects of blood gas analysis during resuscitation

During cardiac arrest, arterial blood gas values are of limited use because they correlate poorly with the severity of hypoxaemia, hypercarbia and acidosis in the tissues. Indeed, during cardiac arrest, venous blood gases may reflect more accurately the acid-base state of the tissues. These are interpreted using the same 5-step approach, however, the normal range of values will be different to arterial blood and they should be interpreted cautiously.

Once return of spontaneous circulation (ROSC) is achieved, arterial blood gas analysis will provide a useful guide to post cardiac arrest treatment, such as the optimal fractional inspired oxygen (FiO_2) and minute ventilation. Arterial lactate concentration can also be used to indicate adequacy of tissue oxygenation, normal arterial blood lactate concentration being 0.7-1.8 mmol l^{-1}. Immediately after cardiac arrest, concentrations are high, reflecting the lactic acidosis that has been caused by inadequate oxygenation of the tissues during the period of cardiac arrest. After ROSC a progressively decreasing lactate value is another indicator of adequate tissue oxygenation.

In the peri-arrest setting, it may be easiest to obtain a sample of arterial blood (into a heparinised syringe) from the femoral artery. The radial artery may be preferable once the patient has an adequate cardiac output and blood pressure. The radial artery is also the best site for insertion of an arterial cannula; this enables continuous monitoring of blood pressure and frequent blood sampling in the post cardiac arrest period.

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10. Pulse oximetry

10.1. Role

Pulse oximetry is a vital adjunct to the assessment of hypoxaemia. Clinical recognition of decreased arterial oxygen saturation of haemoglobin (SaO_2) is subjective and unreliable, with the classic sign of cyanosis appearing late when arterial oxygen saturation is between 80-85%. Pulse oximetry is simple to use, relatively cheap, non-invasive and provides an immediate, objective measure of arterial blood oxygen saturation. It is now used widely in all in-hospital settings and increasingly in both primary care and the pre-hospital environment. Oxygen saturation, 'the fifth vital sign', now also forms a component of many early warning systems to identify the deteriorating patient.

10.2. Principles

The pulse oximeter probe containing light-emitting diodes (LEDs) and a photoreceptor situated opposite, is placed across tissue, usually a finger or earlobe. Some of the light is transmitted through the tissues while some is absorbed. The ratio of transmitted to absorbed light is used to generate the peripheral arterial oxygen saturation (SpO_2) displayed as a digital reading, waveform, or both. As a result of rapid sampling of the light signal, the displayed reading will alter every 0.5-1 s, displaying the average SpO_2 over the preceding 5-10 s. Most pulse oximeters are accurate to within $\pm 2\%$ above an SaO_2 of 90%.

Tissue thickness should be optimally between 5-10 mm. Poor readings may be improved by trying different sites, warming sites or applying local vasodilators.

Pulse oximeters often provide an audible tone related to the SpO_2, with a decreasing tone reflecting increasing degrees of hypoxaemia. Information about pulse rate and waveform (plethysmographic waveform) may also be provided. A poor signal may indicate a low blood pressure or poor tissue perfusion - reassess the patient.

Pulse oximetry provides only a measure of oxygen saturation, not content, and thus gives no indication of actual tissue oxygenation. Furthermore, it provides no information on the partial pressure of carbon dioxide in the body ($PaCO_2$) and is not a measure of adequacy of ventilation. In cases of critical illness, or when type II respiratory failure *(see below)* is suspected (e.g. known COPD, congenital heart disease) arterial blood gas sampling must be performed. Readings from a pulse oximeter must not be used in isolation: it is vital to interpret them in light of the clinical picture and alongside other investigations, and potential sources of error.

10.3. Limitations

The relationship between oxygen saturation and arterial oxygen partial pressure (PaO_2) is demonstrated by the oxyhaemoglobin dissociation curve *(figure 15.1)*. The sinusoid shape of the curve means that an initial decrease from a normal PaO_2 is not accompanied by a drop of similar magnitude in the oxygen saturation of the blood, and early hypoxaemia

may be masked. At the point where the SpO_2 reaches 90-92 %, the PaO_2 will have decreased to around 8 kPa. In other words, the partial pressure of oxygen in the arterial blood will have decreased by almost 50 % despite a reduction in oxygen saturation of only 6-8 %.

The output from a pulse oximeter relies on a comparison between current signal output and standardised reference data derived from healthy volunteers. Readings provided are thus limited by the scope of the population included in these studies, and become increasingly unreliable with increasing hypoxaemia. Below 70 % the displayed values are highly unreliable.

There are several acknowledged sources of error with pulse oximetry:

- presence of other haemoglobins: carboxyhaemoglobin (carbon monoxide poisoning), methaemoglobin (congenital or acquired), fetal haemoglobins and sickling red cells (sickle cell disease)

- surgical and imaging dyes: methylene blue, indocyanine green and indigo carmine cause falsely low saturation readings

- nail varnish (especially blue, black and green)

- high-ambient light levels (fluorescent and xenon lamps)

- motion artefact

- reduced pulse volume:

    - hypotension

    - low cardiac output

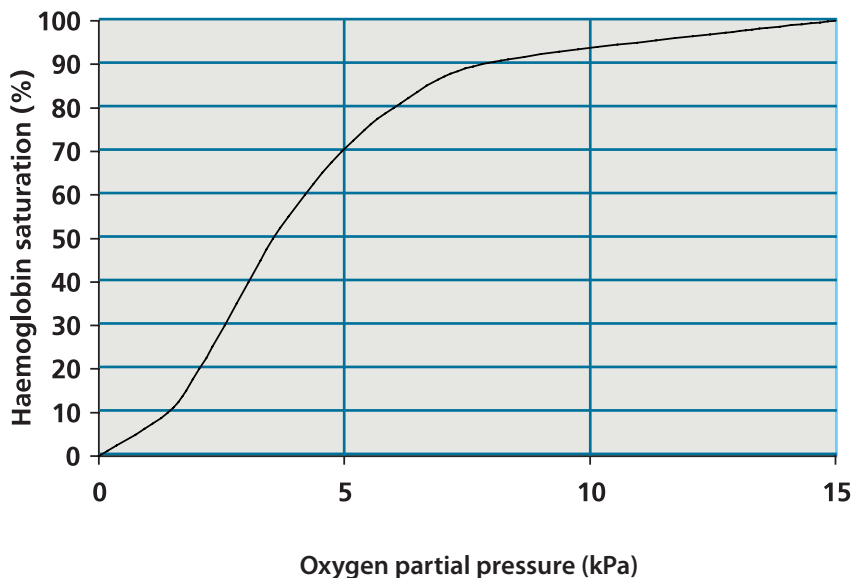   - vasoconstriction

    - hypothermia

Pulse oximeters are not affected by:

- anaemia (reduced haemoglobin)

- jaundice (hyperbilirubinaemia)

- skin pigmentation

Pulse oximetry does not provide a reliable signal during CPR.

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Figure 15.1
Oxyhaemoglobin dissociation curve

Oxygen dissociation curve

Oxygen partial pressure (kPa)

10.4. Uses

Pulse oximetry has four main uses:

1. detection of/screening for hypoxaemia

2. targeting oxygen therapy

3. routine monitoring during anaesthesia

4. diagnostic (e.g. sleep apnoea)

11. Targeted oxygen therapy

In critically ill patients, those presenting with acute hypoxaemia (initial SpO_2 < 85 %), or in the peri-arrest situation, give high-concentration oxygen immediately. Give this initially with an oxygen mask and reservoir ('non-rebreathing' mask) and an oxygen flow of 15 l min^{-1}. During cardiac arrest use 100 % inspired oxygen concentration to maximise arterial oxygen content and delivery to the tissues.

Once ROSC has been achieved and the oxygen saturation of arterial blood can be monitored reliably, adjust the inspired oxygen concentration to maintain a SpO_2 of 94-98 %. If pulse

oximetry (with a reliable reading) is unavailable, continue oxygen via a reservoir mask until definitive monitoring or assessment of oxygenation is available. All critically ill patients will need arterial blood gas sampling and analysis as soon as possible. Evidence suggests both hypoxaemia and hyperoxaemia ($PaO_2 > 20$ kPa) in the post-resuscitation phase may lead to worse outcomes than those in whom normoxaemia is maintained.

11.1. Special clinical situations

Patients with respiratory failure can be divided into two groups:

- **Type I:** low PaO_2 (< 8 kPa), normal $PaCO_2$ (< 6-7 kPa). In these patients it is safe to give a high concentration of oxygen initially with the aim of returning their PaO_2 to normal and then once clinically stable, adjusting the inspired oxygen concentration to maintain an SpO_2 of 94-98 %.

- **Type II:** low PaO_2 (< 8 kPa), increased $PaCO_2$ (> 6-7 kPa). This is often described as hypercapnic respiratory failure and is usually caused by COPD. If given excessive oxygen, these patients may develop worsening respiratory failure with further increases in $PaCO_2$ and the development of a respiratory acidosis. If unchecked, this will eventually lead to unconsciousness, and respiratory and cardiac arrest. The target oxygen saturation in this at risk population should be 88-92 %. However, when critically ill, give these patients high-flow oxygen initially; then analyse the arterial blood gases and use the results to adjust the inspired oxygen concentration. When clinically stable and a reliable pulse oximetry reading is obtained, adjust the inspired oxygen concentration to maintain an SpO_2 of 88-92 %.

In patients with a myocardial infarction or an acute coronary syndrome, and who are not critically or seriously ill, aim to maintain an SpO_2 of 94-98 % (or 88-92 % if the patient is at risk of hypercapnic respiratory failure). This may be achievable without supplementary oxygen, and represents a change from previously accepted practice.

KEY LEARNING POINTS

- The results of arterial blood gas analysis should be interpreted systematically using the 5-step approach.

- Pulse oximetry enables arterial blood oxygen saturation to be monitored continuously.

- During CPR use an inspired oxygen concentration of 100 % until return of spontaneous circulation (ROSC) is achieved.

- After ROSC is achieved, and once the SpO_2 can be monitored reliably, titrate the inspired oxygen concentration to keep the SpO_2 in the range 94-98 % (or 88-92 % in patients at risk of hypercapnic respiratory failure).

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FURTHER READING

- A Simple Guide to Blood Gas Analysis. Eds. Driscoll P, Brown T, Gwinnutt C, Wardle T. BMJ Publishing Group. London 1997.
- O'Driscoll BR, Howard LS, Davison AG. BTS guideline for emergency oxygen use in adult patients. Thorax 2008;63 Suppl 6:vi1-68.

Chapter 16.

Decisions relating to resuscitation

LEARNING OUTCOMES

To understand:

- ethical principles
- advance decisions to refuse treatment
- when not to start cardiopulmonary resuscitation (CPR)
- discussing CPR decisions with patients and those close to them
- who should make decisions about CPR
- when to stop resuscitation attempts

1.   Introduction

Successful resuscitation attempts have brought extended, useful and precious life to many individuals. However, only a minority of people survive and make a complete recovery after attempted resuscitation from cardiac arrest. Attempted resuscitation carries a risk of causing suffering and prolonging the process of dying. It is not an appropriate goal of medicine to prolong life at all costs. Ideally, decisions about whether or not it is appropriate to start cardiopulmonary resuscitation (CPR) should be made in advance, as part of the overall concept of advance care planning. Detailed guidance has been published on a national basis in most European countries. As an ALS provider, you should read and be familiar with that guidance and follow the principles that it contains.

It is incumbent on all healthcare practitioners to practice within the law. The law as it relates to CPR varies from country to country. Even within one nation there are some differences between countries. As an ALS provider you should be familiar with the relevant aspects of law in the country where you live and work.

Discussing decisions about CPR can be difficult and distressing for patients and relatives, and for healthcare providers. These decisions may be influenced by various factors including personal beliefs and opinions, cultural or religious influences, ethical and legal considerations, and by social or economic circumstances. Some patients with capacity

decide that they do not want treatment and record their wishes in an advance decision to refuse treatment (formerly known as 'living wills'). As an ALS provider you should understand the ethical and legal principles as well as the clinical aspects involved before undertaking discussions or making a decision about CPR.

2.   Principles

The four key principles of medical ethics are summarised in the box:

- **Beneficence** requires provision of benefit while balancing benefit and risks. Commonly this will involve attempting CPR but if risks clearly outweigh any likely benefit it will mean withholding CPR. Beneficence includes also responding to the overall needs of the community, such as establishing a programme of public access defibrillation.

- **Non-maleficence** means doing no harm. CPR should not be attempted in people in whom it will not succeed, where no benefit is likely but there is a clear risk of harm.

- **Justice** implies a duty to spread benefits and risks equally within a society. If CPR is provided, it should be available to all who may benefit from it; there should be no discrimination purely on the grounds of factors such as age or disability.

- Autonomy relates to people making their own informed decisions rather than healthcare professionals making decisions for them. Autonomy requires that a person with capacity is adequately informed, is free from undue pressure, and that there is consistency in their preferences.

3.   Advance decisions to refuse CPR

Advance decisions to refuse treatment have been introduced in many countries and emphasise the importance of patient autonomy. Resuscitation must not be attempted if CPR is contrary to the recorded, sustained wishes of an adult who had capacity and was aware of the implications at the time of making that advance decision. However, it is important to ensure that an advance decision is valid and that the circumstances in which the decision is applied are those that were envisaged or defined at the time that it was made.

The term 'advance decision' may apply to any expression of patient preferences. Refusal does not have to be in writing in order to be valid. If patients have expressed clear and consistent refusal verbally, this is likely to have the same status as a written advance decision. People should ensure that their healthcare team and those close to them are aware of their wishes.

In sudden out-of-hospital cardiac arrest, those attending usually do not know the patient's situation and wishes and, even if an advance decision has been recorded, it may not be available. In these circumstances CPR can be started immediately and any further information obtained when possible. There is no ethical difficulty in stopping a resuscitation attempt that has started if the healthcare professionals are presented later with a valid advance decision refusing the treatment that has been started.

There is still considerable international variation in the medical attitude to written advance decisions. In some countries, such as the UK, a written advance decision is legally binding. Where no explicit advance decision has been made and the express wishes of the patient are unknown there is a presumption that healthcare professionals will, if appropriate, make all reasonable efforts to resuscitate the patient.

4.   When to withhold CPR

While patients have a right to refuse treatment, they do not have an automatic right to demand treatment; they cannot insist that resuscitation must be attempted in any circumstance. Doctors cannot be required to give treatment that is contrary to their clinical judgement. This type of decision is often complex and should be undertaken by senior, experienced members of the medical team.

The decision to make no resuscitation attempt raises several ethical and moral questions. What constitutes futility? What exactly should be withheld? Who should decide and who should be consulted? Who should be informed?

4.1.   What constitutes futility?

Futility may be considered to exist if resuscitation will not prolong life of a quality that would be acceptable to the patient. Although predictors of non-survival after attempted resuscitation have been published, none has sufficient predictive value when applied to an independent validation group. Furthermore, the outcome for a cohort undergoing attempted resuscitation is dependent on system factors such as time to CPR and time to defibrillation. It is difficult to predict how these factors will impact on the outcome of individuals.

Inevitably, judgements will have to be made, and there will be grey areas where subjective opinions are required in patients with comorbidity such as heart failure, chronic respiratory disease, asphyxia, major trauma, head injury and neurological disease. The age of the patient may feature in the decision but is only a relatively weak independent predictor of outcome; however, the elderly commonly have significant comorbidity, which influences outcome.

4.2.   What exactly should be withheld?

Do not attempt resuscitation (DNAR) means that in the event of cardiac or respiratory arrest, CPR should not be started - nothing more than that. Other treatment should be continued, including pain relief and sedation, as required. Treatment such as ventilation and oxygen therapy, nutrition, antibiotics, fluid and vasopressors, is also continued as indicated. If not, orders not to continue or initiate any such treatments should be made independently of DNAR orders.

In the past, in many countries, doctors would make a DNAR decision without consulting with the patient, the relatives, or other members of the health care team. Many countries have now published clear guidelines on how these decisions should be taken. In most cases, this guidance emphasises involvement by the patient and/or relatives.

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4.3. Who should decide not to attempt resuscitation and who should be consulted?

The overall responsibility for this decision rests with the senior healthcare professional in charge of the patient after appropriate consultation with other healthcare professionals involved in the patient's care.

People have ethical and legal rights to be involved in decisions that relate to them and if the patient has capacity their views should be sought unless there is a clearly justifiable reason to indicate otherwise. It is not necessary to initiate discussion about CPR with every patient, for example if there is no reason to expect cardiac arrest to occur, or if the patient is in the final stage of an irreversible illness in which CPR would be inappropriate as it would offer no benefit.

It is good practice to involve relatives in decisions although they have no legal status in terms of actual decision-making. A patient with capacity should give their consent before involving the family in a DNAR discussion. Refusal from a patient with capacity to allow information to be disclosed to relatives must be respected.

If patients who lack capacity have previously appointed a welfare attorney with power to make such decisions on their behalf, that person must be consulted when a decision has to be made balancing the risks and burdens of CPR.

In some circumstances there are legal requirements to involve others in the decision-making process when a patient lacks capacity. For example the Mental Capacity Act 2005, which applies in England and Wales requires appointment of an Independent Mental Capacity Advocate (IMCA) to act on behalf of the patient who lacks capacity. However, when decisions have to be made in an emergency, there may not be time to appoint and contact an IMCA and decisions must be made in the patient's best interests, and the basis for such decisions documented clearly and fully.

When differences of opinion occur between the healthcare team and the patient or their representatives these can usually be resolved with careful discussion and explanation, or if necessary by obtaining a second clinical opinion. In general, decisions by legal authorities are often fraught with delays and uncertainties, especially if there is an adversarial legal system, and formal legal judgement should be sought only if there are irreconcilable differences between the parties involved. In particularly difficult cases, the senior doctor may wish to consult his/her own medical defence society for a legal opinion.

4.4. Who should be informed?

Once the decision has been made it must be communicated clearly to all who may be involved, including the patient. Unless the patient refuses, the decision should also be communicated to the patient's relatives. The decision, the reasons for it, and a record of who has been involved in the discussions should be recorded in the medical notes - ideally on a special DNAR form - and should clearly document the date the decision was made. The decision should be recorded in the nursing records, if these are separate. The decision must be communicated to all those involved in the patient's care.

5. Communicating decisions about CPR to patients and those close to them

Whilst it is generally advisable to explain to patients and those close to them any decisions that have been taken about their treatment, and the reasons for those decisions, it is important that this is not done without careful consideration. On the other hand, it may sometimes not be necessary to inform every patient about a decision not to attempt CPR because it would not be successful, discussing that decision would be unnecessarily distressing and of little or no value to the patient. Any discussion with those close to patients must respect the patient's wishes in relation to confidentiality.

6. Communicating decisions about CPR to the healthcare team

Good communication within the team is an essential component of high-quality, safe healthcare. When a decision is made not to attempt CPR, the basis for that decision, details of those involved in making it, and details of discussions with patients and those close to them should be recorded. The decision itself should be recorded in a way that is immediately available and recognisable to those present, should the patient suffer sudden cardiac arrest. Most European countries have defined standards for the recording of decisions relating to CPR. Such decisions were referred to at one time as 'Do Not Resuscitate' (DNR) decisions. DNR was replaced by DNAR ('Do Not Attempt Resuscitation') to emphasise the reality that many resuscitation attempts will not be successful. Unfortunately some healthcare providers have mistakenly and inappropriately interpreted the recording of these decisions as indicating that other treatment can or should be withheld. To discourage this it has been suggested that the term DNACPR ('Do Not Attempt CPR') should be used, to try to emphasise that the recorded decision refers only to the use of CPR and not to any other aspect of treatment that the patient may need. As an ALS provider you should ensure that you record decisions about CPR fully, clearly and accurately, and that these decisions do not (through your actions or those of others) lead to withholding from patients other treatment that they may need. Whilst the term 'DNAR' is used throughout ERC material it is interchangeable and identical in definition with the term 'DNACPR' which is also in common use.

7. When to stop CPR

Most of resuscitation attempts do not succeed and in those that are unsuccessful a decision has to be made to stop CPR. This decision can be made when it is clear that continuing CPR will not be successful. Factors influencing the decision will include the patient's medical history and prognosis, the cardiac arrest rhythm that is present, the response or lack of response to initial resuscitation measures, and the duration of the resuscitation attempt (particularly if the rhythm is asystole - *see below*). Sometimes, during a resuscitation attempt, further information becomes available that was not known at the time CPR was started, and that indicates that further CPR will not succeed. It is appropriate to stop CPR in those circumstances.

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In general, CPR should be continued as long as a shockable rhythm or other reversible cause for cardiac arrest persists. It is generally accepted that asystole for more than 20 min in the absence of a reversible cause *(see below)*, and with all advanced life support measures in place, is unlikely to respond to further CPR and is a reasonable basis for stopping CPR.

A decision to abandon CPR is made by the team leader, but this should be after consultation with the other team members. Ultimately, the decision is based on a clinical judgement that further advanced life support will not re-start the heart and breathing.

8. Decision making by non-doctors

Many cases of out-of-hospital cardiac arrest are attended by emergency medical technicians or paramedics, who face similar dilemmas about when CPR will not succeed and when it should be stopped. In general CPR will be started in out-of-hospital cardiac arrest unless there is a valid advance decision refusing it or a valid DNAR order or it is clear that CPR would be futile, for example, in cases of mortal injuries such as decapitation or hemicorporectomy, known prolonged submersion, incineration, rigor mortis, and dependent lividity. In such cases, the non-doctor can identify that death has occurred but does not certify the cause of death (which in most countries can be done only by a physician or coroner).

But when should a decision be made to abandon a resuscitation attempt? For example, should ALS trained paramedics be able to declare death when the patient remains in asystole after 20 min despite ALS interventions? In some countries, including the UK, paramedics may cease a resuscitation attempt in this situation. Their strict protocol requires that certain conditions that might indicate a remote chance of survival (e.g. hypothermia) are absent. The presence of asystole must also be established beyond reasonable doubt and documented on ECG recordings *(see chapter 14)*.

Similar decisions about initiating resuscitation or recognising that death has occurred and is irreversible may be made by experienced nurses, working in the community or in establishments that provide care for people who are terminally or chronically ill. Whenever possible in such settings, decisions about CPR should be considered before they are needed, as part of advance care planning. In some situations it will be appropriate for experienced nurses to undertake any necessary discussions and to make and record a DNAR order on behalf of the patient and their healthcare team.

9. Special circumstances

Certain circumstances, e.g. hypothermia at the time of cardiac arrest, will enhance the chances of recovery without neurological damage. In such situations do not use the usual prognostic criteria (such as asystole persisting for more than 20 min) and continue CPR until the reversible problem has been corrected (e.g. re warming has been achieved).

10. Withdrawal of other treatment after a resuscitation attempt

Prediction of the likely clinical and neurological outcome in people who remain unconscious after regaining a spontaneous circulation is difficult during the first 3 days. In general, other supportive treatment should be continued during this period, after which the prognosis can be assessed with greater confidence. This topic is covered in more detail in chapter 13.

KEY LEARNING POINTS

- In the event of cardiac arrest, CPR should be started promptly and effectively.

- If a valid advance decision refusing CPR has been made, do not attempt CPR.

- When CPR will not re-start the heart and breathing, CPR is not appropriate.

- If continuing CPR will not be successful, make the decision to stop.

- Decisions relating to CPR should be made carefully, recorded fully, and communicated effectively.

- Decisions relating to CPR should not prevent patients from receiving any other treatment needed.

FURTHER READING

- Bossaert L et al. European Resuscitation Council Guidelines for Resuscitation 2015
- Section 11. The ethics of resuscitation and end-of-life decisions. 10.1016/j.resuscitation.2015.07.033; p301 - p310
- Baskett PJ, Lim A. The varying ethical attitudes towards resuscitation in Europe. Resuscitation 2004;62:267-73.
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Chapter 17.

Supporting the relative in resuscitation practice

> **LEARNING OUTCOMES**
>
> **To understand:**
> - how to support relatives witnessing attempted resuscitation
> - how to care for the recently bereaved
> - religious and cultural requirements when a patient has died
> - the legal and practical arrangements following a recent death

Throughout this chapter, the term 'relatives' includes close friends/significant others.

1.   Introduction

In many cases of out-of-hospital cardiac arrest, the person who performs CPR will be a close friend or relative and they may wish to remain with the patient.

Many relatives find it more distressing to be separated from their family member during these critical moments than to witness attempts at resuscitation. In keeping with the move to more open clinical practice, healthcare professionals should take the preferences of patients and relatives into account.

If the resuscitation attempt fails, relatives perceive a number of advantages of being present during resuscitation:

- It helps them come to terms with the reality of death, avoiding prolonged denial and contributing to a healthier bereavement.
- The relative can speak while there is still a chance that the dying person can hear.
- They are not distressed by being separated from a loved one at a time when they feel the need to be present.
- They can see that everything possible was done for the dying person, which assists with their understanding of the reality of the situation.
- They can touch and speak with the deceased whilst the body is warm.

There are potential disadvantages of relatives being present:

- The resuscitation attempt may prove distressing, particularly if the relatives are not kept informed.

- Relatives can physically, or emotionally, hinder the staff involved in the resuscitation attempt. Observed actions or remarks by medical or nursing staff may offend grieving family members.

- Relatives may be disturbed by the memory of events, although evidence indicates that fantasy is worse than fact. The staff should take into account the expectations of the bereaved and their cultural background during and following death.

- Relatives may demonstrate their emotions vocally or physically whilst others may wish to sit quietly or read religious text. The staff must have sufficient insight, knowledge and skills to anticipate these needs and identify potential problems.

2.   Caring for the recently bereaved

Care and consideration of the relative during resuscitation becomes increasingly important as procedures become more invasive. Support should be provided by an appropriately qualified healthcare professional whose responsibility is to care for family members witnessing cardiopulmonary resuscitation. The following safeguards should be used:

- Acknowledge the difficulty of the situation. Ensure that they understand that they have a choice of whether or not to be present during resuscitation. Avoid provoking feelings of guilt whatever their decision.

- Explain that they will be looked after whether or not they decide to witness the resuscitation attempt. Ensure that introductions are made and names are known.

- Give a clear explanation of what has happened in terms of the illness or injury and what they can expect to see when they enter the resuscitation area.

- Ensure that the relatives understand that they will be able to leave and return at any time, and will always be accompanied.

- Ask the relative not to interfere with the resuscitation process but offer them the opportunity to touch the patient when they are told that it is safe to do so.

- Explain the nature of the procedures in simple terms. If resuscitation is unsuccessful, explain why the attempt has been stopped.

If the patient dies, advise the relatives that there may be a brief interval while equipment is removed, after which they can return to be together in private. Under some circumstances, the coroner may require certain equipment to be left in place. Offer the relatives time to think about what has happened and the opportunity for further questions.

Hospitals should develop policies to enable relatives to observe the attempted resuscitation of their loved one.

3.   Caring for the recently bereaved

Caring for the bereaved compassionately will ease the grieving process. Adapt the following considerations to the individual family and their cultural needs:

- early contact with one person, usually a nurse

- provision of a suitable area for the relatives to wait, e.g. relatives' room

- breaking bad news sympathetically and supporting the grief response appropriately

- arranging for relatives to view the body

- religious and pastoral care requirements

- legal and practical arrangements

- follow up and team support

3.1.   Early contact with one person

Ideally this should be the person who has supported the relatives during the resuscitation attempt. If the resuscitation attempt was not observed allocate a member of the care team specifically to support the relatives. Communication between the emergency services and the receiving hospital should ensure that the arrival of relatives is anticipated for an out-of-hospital arrest. A warm, friendly and confident greeting will help to establish an open and honest relationship.

3.2.   Provision of a suitable room

This should provide the appropriate ambience, space and privacy for relatives to ask questions and to express their emotions freely.

3.3.   Breaking bad news and supporting the grief response

An uncomplicated and honest approach will help avoid mixed messages. The most appropriate person (not necessarily a doctor) should break the bad news to the relatives. It may be more appropriate for the nurse who has been accompanying the relatives to break the news, although relatives may take comfort from talking to a doctor as well and this opportunity should always be offered. When preparing to talk to the relatives, consider the following:

- Prepare yourself mentally and physically. Check your clothing for blood, wash your hands and tidy your clothing.

- Confirm that you are talking with the correct relatives and establish their relationship to the deceased. Briefly establish what they know and use this as the basis for your communication with them.

- Use tone of voice and non-verbal behaviour to support what you are saying. Smiles, nods, eye contact, the use of touch, facial expression and gestures can help support verbal communication.

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- Use simple words and avoid medical jargon and platitudes that will be meaningless to the relative.

- Sit or position yourself next to the relative so that you are on the same level.

- Do not enter into a long preamble or start to question the relative about issues such as premorbid health. They want to know immediately whether their loved one is alive or dead.

- Introduce the word "dead", "died" or "death" at the earliest moment and reinforce this on at least one further occasion, so that there is no ambiguity.

- After breaking the news, do not be afraid to allow a period of silence while the facts are absorbed.

- Anticipate the different types of reaction/emotional response you may experience after breaking the bad news.

3.4. Possible responses to grief include:

- acute emotional distress/shock

- anger

- denial/disbelief

- guilt

- catatony

These stages are not linear and individuals may move from one to another, returning to some repeatedly. An individual's gender, age and cultural background will influence the response to grief. Respect cultural requirements and, where possible, provide written guidelines for individual ethnic groups.

3.5. Arranging viewing of the body

Many newly bereaved relatives value the opportunity to view their loved ones. Their experience is likely to be affected by whether the deceased appears in a presentable condition. Advise relatives what to expect before viewing the body. People are less concerned about medical devices and equipment than is generally believed. If the deceased has mutilating injuries, warn the relatives. Being in the physical presence of their loved one will help them work through the grieving process. Ensure the opportunity to touch/hold the deceased is given. Staff should accompany relatives during the viewing process and they should remain near by to offer support or provide information as required.

3.6. Religious requirements, legal and practical arrangements

Variations in handling the body and expressions of grief are influenced by the patient's religious convictions. The resuscitation team should take into account the beliefs, values and rituals of the patient and the family. There is an increasing emphasis on the need for care practice to be culturally sensitive, as a way of valuing and respecting the cultural and

religious needs of patients. Religious representatives from the patient's denomination or faith are usually available to attend in hospital. Hospital chaplains are a great source of strength and information to families and staff. Prayers, blessings, religious acts and procedure are all important in ensuring that relatives are not distressed further.

Legal and practical arrangements are equally important. These include:

- notification of the coroner or other appropriate authority
- notification of the patient's family doctor
- organ donation decisions
- provision of information about what to do in the event of death
- involvement of religious ministers
- adhere to hospital procedure in the return of patients property and valuables
- information concerning the social services that are available
- information concerning post mortem examination where indicated
- follow-up arrangements, which may involve long-term counselling
- provision of a telephone contact number for relatives to use and a named staff member who they can call should they have any further questions

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KEY LEARNING POINTS

- Many relatives want the opportunity to be present during the attempted resuscitation of their loved one. This may help the grieving process.

- Communication with bereaved relatives should be honest, simple, and supportive.

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FURTHER READING

- Bossaert L, Perkins GD, Askitopoulou H, et al. European Resuscitation Council Guidelines for Resuscitation 2015 Section 11 The Ethics of Resuscitation and End-of-Life Decisions. 10.1016/j.resuscitation.2015.07.033; p301 - p310

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Drugs used in the treatment of Cardiac Arrest

Drug	Shockable (VF/Pulseless VT)	Non-Shockable (PEA/Asystole)
Adrenaline	• Dose: 1 mg (10 ml 1:10,000 or 1 ml 1:1,000) IV • Given after the 3rd shock if iv/io access is obtained • Repeated every 3-5 min (alternate loops) • Give without interrupting chest compressions	• Dose: 1 mg (10 ml 1:10,000 or 1 ml 1:1,000) IV • Given as soon as iv/io access is obtained • Repeated every 3-5 min (alternate loops) • Give without interrupting chest compressions
	Adrenaline has been the primary sympathomimetic drug for the management of cardiac arrest for 45 years. Its alpha-adrenergic effects cause systemic vasoconstriction, which increases macrovascular coronary and cerebral perfusion pressures. The beta-adrenergic actions of adrenaline (inotropic, chronotropic) may increase coronary and cerebral blood flow, but concomitant increases in myocardial oxygen consumption and ectopic ventricular arrhythmias (particularly in the presence of acidaemia), transient hypoxaemia because of pulmonary arteriovenous shunting, impaired microcirculation, and increased post cardiac arrest myocardial dysfunction may offset these benefits. Adrenaline use is associated with more rhythm transitions during ALS, both during VF and PEA. Although there is no evidence of long-term benefit from the use of adrenaline, the improved short-term survival documented in some studies warrants its continued use.	
Amiodarone	• Dose: 300 mg bolus IV • Given after the 3rd shock if iv/io access is obtained • Further dose of 150 mg after the 5th shock if VF/pVT persists	• Not indicated for PEA or asystole
	Amiodarone is a membrane-stabilising anti-arrhythmic drug that increases the duration of the action potential and refractory period in atrial and ventricular myocardium. Atrioventricular conduction is slowed, and a similar effect is seen with accessory pathways. Amiodarone has a mild negative inotropic action and causes peripheral vasodilation through non-competitive alpha-blocking effects. The hypotension that occurs with intravenous amiodarone is related to the rate of delivery and is caused by the solvent, rather than the drug itself. When amiodarone is unavailable, consider an initial dose of 100 mg (1-1.5 mg kg^{-1}) of lidocaine for VF/pVT refractory to three shocks. Give an additional bolus of 50 mg if necessary. The total dose should not exceed 3 mg kg^{-1} during the first hour.	

Drug	Shockable (VF/Pulseless VT)	Non-Shockable (PEA/Asystole)
Magnesium	• Dose: 2 g given peripherally IV • May be repeated after 10-15 min • Indicated for VT, torsade de pointes, or digoxin toxicity associated with hypomagnesaemia	• Dose: 2 g given peripherally IV • May be repeated after 10-15 min • Indicated for supraventricular tachycardia or digoxin toxicity associated with hypomagnesaemia
	Magnesium facilitates neurochemical transmission: it decreases acetylcholine release and reduces the sensitivity of the motor endplate. Magnesium also improves the contractile response of the stunned myocardium, and may limit infarct size.	
Calcium	• Not indicated for shockable rhythms	• Dose: 10 ml 10 % calcium chloride (6.8 mmol Ca^{2+}) IV • Indicated for PEA caused specifically by hyperkalaemia, hypocalcaemia or overdose of calcium channel blocking drugs
	Calcium plays a vital role in the cellular mechanisms underlying myocardial contraction. High plasma concentrations achieved after injection may be harmful to the ischaemic myocardium and may impair cerebral recovery. Do not give calcium solutions and sodium bicarbonate simultaneously by the same route.	

Sodium Bicarbonate	• Dose: 50 mmol (50 ml of an 8.4 % solution) IV
	• Routine use not recommended
	• Consider sodium bicarbonate in shockable and non-shockable rhythms for
	    - cardiac arrest associated with hyperkalaemia
	    - tricyclic overdose.
	Repeat the dose as necessary, but use acid-base analysis to guide therapy.	

	Cardiac arrest results in combined respiratory and metabolic acidosis because pulmonary gas exchange ceases and cellular metabolism becomes anaerobic. The best treatment of acidaemia in cardiac arrest is chest compression; some additional benefit is gained by ventilation. Bicarbonate causes generation of carbon dioxide, which diffuses rapidly into cells. This has the following effects: • it exacerbates intracellular acidosis • it produces a negative inotropic effect on ischaemic myocardium • it presents a large, osmotically-active sodium load to an already compromised circulation and brain • it produces a shift to the left in the oxygen dissociation curve, further inhibiting release of oxygen to the tissues Do not give calcium solutions and sodium bicarbonate simultaneously by the same route.	
Fluids	Infuse fluids rapidly if hypovolaemia is suspected. During resuscitation, there are no clear advantages in using colloid, so use 0.9 % sodium chloride or Hartmann's solution. Avoid dextrose, which is redistributed away from the intravascular space rapidly and causes hyperglycaemia, which may worsen neurological outcome after cardiac arrest.	

Drug	Shockable (VF/Pulseless VT)	Non-Shockable (PEA/Asystole)
Thrombolytics	• Tenecteplase 500-600 mcg kg^{-1} IV bolus • Alteplase (r-tPA) 6 mg kg^{-1} IV bolus	
	Fibrinolytic therapy should not be used routinely in cardiac arrest. Consider fibrinolytic therapy when cardiac arrest is caused by proven or suspected acute pulmonary embolus. If a fibrinolytic drug is given in these circumstances, consider performing CPR for at least 60-90 min before termination of resuscitation attempts. Ongoing CPR is not a contraindication to fibrinolysis.	

APPENDIX B

Drugs used in the peri-arrest period

Drug	Indication	Dose
Adenosine	• Paroxysmal SVT with re-entrant circuits that include the atrioventricular (AV) node (AVNRT and AVRT)	• 6 mg IV bolus (in some countries only 5 mg preparations available) • If unsuccessful, give up to two doses of 12 (10) mg after 1-2 min intervals
	Adenosine is a naturally occurring purine nucleotide. It slows transmission through the AV node but has little effect on other myocardial cells or conduction pathways. It is highly effective for terminating paroxysmal SVT with re-entrant circuits that include the AV node (AVNRT). In other narrow-complex tachycardias, adenosine will reveal the underlying atrial rhythms by slowing the ventricular response. It has an extremely short half-life of 10-15 s and, therefore, is given as a rapid bolus into a fast running intravenous infusion or followed by a saline flush. Warn patients of transient unpleasant side effects; in particular, nausea, flushing, and chest discomfort. It is contraindicated in patients with known or suspected bronchoconstrictive or bronchospastic lung disease.	
Adrenaline	• Second-line treatment for cardiogenic shock • Bradycardia (alternative to external pacing) • Anaphylaxis	• 0.05-1 mcg kg^{-1} min^{-1} • 2-10 mcg min^{-1} • *See chapter 12*
	An adrenaline infusion is indicated in the post-resuscitation period when less potent inotropic drugs (e.g. dobutamine) have failed to increase cardiac output adequately. It is indicated also for bradycardia associated with adverse signs and/or risk of asystole, which has not responded to atropine, if external pacing is unavailable or unsuccessful.	
Amiodarone	• Control of haemodynamically stable monomorphic VT, polymorphic VT and wide-complex tachycardia of uncertain origin • Paroxysmal SVT uncontrolled by adenosine, vagal manoeuvres or AV nodal blockade • To control a rapid ventricular rate caused by accessory pathway conduction in pre-excited atrial arrhythmias In patients with pre-excitation and AF, digoxin, non-dihydropyridine calcium channel antagonists, or intravenous amiodarone should not be administered as they may increase the ventricular response and may result in VF	• 300 mg IV over 1-60 min (depending on haemodynamic stability of patient) • Followed by 900 mg over 24 h • Additional infusions of 150 mg can be repeated as necessary for recurrent or resistant arrhythmias to a maximum manufacturer-recommended total daily dose of 2 g (this maximum licensed dose varies between different countries)

Drug	Indication	Dose
Amiodarone	• After unsuccessful electrical cardioversion, to achieve chemical cardioversion or to increase the likelihood of further electrical cardioversion succeeding	
	Intravenous amiodarone has effects on sodium, potassium and calcium channels as well as alpha- and beta-adrenergic blocking properties. In patients with severely impaired heart function, intravenous amiodarone is preferable to other anti-arrhythmic drugs for atrial and ventricular tach-yarrhythmias. Major adverse effects (caused by the solvent, not the active drug) are hypotension and bradycardia, which can be minimised by slowing the rate of drug infusion. Whenever possible, intravenous amiodarone should be given via a central venous catheter; it causes thrombophlebitis when infused into a peripheral vein, but in an emergency it can be injected into a large peripheral vein.	
Acetylsalicyl-ic acid (ASA)	• Acute coronary syndromes	• 150 to 300 mg of a non-enteric coated formulation or 150 mg of an IV preparation as soon as possible
	Aspirin improves the prognosis of patients with acute coronary syndromes, significantly reducing cardiovascular death. The efficacy of aspirin is achieved by anti-platelet activity and preventing early platelet thrombus formation.	
Atropine	• Sinus, atrial, or nodal bradycardia or AV-block, when the haemodynamic condition of the patient is unstable because of the bradycardia.	• 500 mcg IV • Repeated doses to maximum of 3 mg
	Atropine antagonises the action of the parasympathetic neurotransmitter acetylcholine at muscarinic receptors. Therefore, it blocks the effect of the vagus nerve on both the sinoatrial (SA) node and the AV node, increasing sinus automaticity and facilitating AV node conduction. Side effects of atropine are dose-related (blurred vision, dry mouth and urinary retention). It can cause acute confusion, particularly in elderly patients. Asystole during cardiac arrest is usually caused by primary myocardial pathology rather than excessive vagal tone and there is no evidence that routine use of atropine is beneficial in the treatment of asystole or PEA. Use atropine cautiously in the presence of acute coronary ischaemia or myocardial infarction; increased heart rate may worsen ischaemia or in-crease the zone of infarction.	

Drug	Indication	Dose
Beta-adrenoceptor blockers	• Narrow-complex regular tachy-cardias uncontrolled by vagal manoeuvres or adenosine in patients with preserved ventricu-lar function • To control rate in atrial fibrillation (AF) and atrial flutter when ventricular function is preserved.	*Atenolol (beta1)* • 5 mg IV over 5 min, repeated if necessary after 10 min *Metoprolol (beta1)* • 2-5 mg IV at 5-min intervals to a total of 15 mg *Propranolol (beta1 and beta2 effects)* • 100 mcg kg^{-1} IV slowly in three equal doses at 2-3 min intervals *Esmolol* • short-acting (half-life of 2-9 min) beta1- selective beta-blocker • IV loading dose of 500 mcg kg^{-1} over 1 min • followed by a titrated infusion of 50-200 mcg kg^{-1} min^{-1}
	Beta blocking drugs reduce the effects of circulating catecholamines and decrease heart rate and blood pressure. There is no evidence to support routine intravenous beta-blockers in the prehospital or initial ED settings. Side effects of beta blockade include bradycardia, AV conduction delay, hypotension and bronchospasm. Contraindications to the use of beta-adrenoceptor blocking drugs include second- or third-degree heart block, hypotension, severe congestive heart failure and lung disease associated with bronchospasm.	
Verapamil	• Stable regular narrow-complex tachycardias uncontrolled or unconverted by vagal manoeuvres or adenosine • To control ventricular rate in pa-tients with AF or atrial flutter and preserved ventricular function	• 2.5-5 mg intravenously given over 2 min • In the absence of a therapeutic response or drug-induced adverse event, give repeated doses of 5-10 mg every 15-30 min to a maximum of 20 mg.
	Verapamil is a calcium channel blocking drug that slows conduction and increases refractoriness in the AV node. These actions may terminate re-en-trant arrhythmias and control the ventricular response rate in patients with atrial tachycardias (including AF and atrial flutter). Intravenous verapamil should be given only to patients with narrow-complex paroxysmal SVT or arrhythmias known with certainty to be of supraventricular origin. Giving calcium channel blockers to a patient with ventricular tachycardia may cause cardiovascular collapse. Verapamil may decrease myocardial contractility and critically reduce cardiac output in patients with severe LV dysfunction.	

Drug	Indication	Dose
Positive inotropic drugs	• Hypotension in the absence of hypovolaemia • Cardiogenic shock	*Dobutamine* 5-20 mcg kg^{-1} min^{-1} *Dopamine* 1-10 mcg kg^{-1} min^{-1} *Noradrenaline* 0.05-1 mcg kg^{-1} min^{-1}
	Dobutamine is often the positive inotropic drug of choice in the post-resuscitation period. Its beta agonist activity also causes vasodilation and an increase in heart rate. It is indicated when poor cardiac output and hypotension cause significantly reduced tissue perfusion. It is useful particularly when pulmonary oedema is present and hypotension prevents the use of other vasodilators. Dopamine is the precursor of the naturally occurring catecholamines adrenaline and noradrenaline. It has a dose dependent positive inotropic effect. Noradrenaline is a potent vasoconstrictor but also has a positive inotropic effect. It is indicated in the post resuscitation period when hypotension and poor cardiac output cause reduced tissue perfusion.	
Magnesium	• Polymorphic ventricular tachycardia (torsade de pointes) • Digoxin toxicity	• Dose: 2 g given peripherally (IV) over 10 min • May be repeated once if necessary
	Magnesium facilitates neurochemical transmission: it decreases acetylcholine release and reduces the sensitivity of the motor endplate.	
Nitrates	• Prophylaxis or relief of angina • Unstable angina pectoris • Myocardial infarction • Acute and chronic left ventricular failure	• GTN: Sublingual 400 mcg (spray or tablet) every 5 min up to 3 doses; isosorbide mononitrate or dinitrate 30-120 mg oral per day (various preparations and dosing frequencies); transdermal 5-15 mg daily • GTN: Sublingual 300-600 mcg (spray or tablet); buccal tablets 2-5 mg; IV 10-200 mcg min^{-1}; isosorbide mononitrate or dinitrate 30-120 mg oral per day (various preparations and dosing frequencies) • GTN: Sublingual 300-600 mcg (spray or tablet); buccal 2-5 mg; IV 10-200 mcg min^{-1} • GTN: iv begin at 10-200 mcg min^{-1} IV in persistent pain or pulmonary edeme; titrate to desired BP effect isosorbide mononitrate or dinitrate 30-120 mg oral per day (various preparations and dosing frequencies); transdermal 5-15 mg daily
	After conversion to nitric oxide, nitrates cause vascular smooth muscle relaxation. The resultant dilation is more marked on the venous than the arterial side of the circulation, and it is this venodilatation, reducing left ventricular diastolic pressure, that is mainly responsible for relieving angina. Nitrates also dilate the coronary arteries and relieve spasm in coronary smooth muscle. Nitrates are contraindicated in hypotensive patients (systolic blood pressure < 90 mmHg).	

APPENDIX C
Useful websites

www.erc.edu	European Resuscitation Council
www.rescusitation.be	Belgian Resuscitation Council
www.resus.org.uk	Resuscitation Council UK
www.ilcor.org	International Liaison Committee on Resuscitation
www.americanheart.org	American Heart Association
www.ics.ac.uk	Intensive Care Society
www.aagbi.org	Association of Anaesthetists of Great Britain and Ireland
www.bestbets.org	Best evidence topics in emergency medicine
www.bcs.com	British Cardiac Society
www.escardio.org	European Society of Cardiology
www.feel-uk.com	Focused Echocardiography in Emergency Life Support

Personal Notes

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